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HUGHLINGS JACKSON ON APHASIA AND KINDRED AFFECTIONS OF SPEECH

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TOGETHER WITH A COMPLETE BIBLIOGRAPHY OF DR. JACKSON'S PUBLICATIONS ON SPEECH, AND A REPRINT OF SOME OF THE MORE IMPORTANT PAPERS.

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CHAPTER I.—INTRODUCTORY.

It is generally conceded that the views on aphasia and analogous disturbances of speech found in the text-books of to-day are of little help in understanding an actual case of disease. The tendency to appear exact by disregarding the complexity of the factors is an old failing in medical history. Each patient with a speech defect of cerebral origin is stretched on the procrustean bed of some theoretical scheme: something is lopped away at one part, something added at another, until the phenomena are said to correspond to [illegible] diagrammatic conception, which never has and never could have existed. And yet neurologists continue to cling to these schemes, modifying them to suit each case, conscious that they do not correspond in any way to the facts they are supposed to explain.

The ground has been worked over again and again to such an extent that it is difficult for the modern student of neurology to throw himself back to the 'sixties, when the subject of speech defects was virgin soil. But of late years dissatisfaction with the current dogmas, manifested in such a revolt as that of Pierre Marie, has driven more thoughtful neurologists back to the earlier work on aphasia. We have become lost in a maze of subtleties, and in attempting to find a new line of advance have groped our way back to the beginning.

Hughlings Jackson was one of the most remarkable pioneers in the field of defects of speech caused by cerebral disease. Between 1864 and 1890 he published a series of papers on this subject, and throughout the whole of this period he would enunciate his views to anyone who visited the wards of the National Hospital. But even amongst the younger men his aphoristic dicta fell upon deaf ears. Until a year ago, when Arnold Pick dedicated "Die agrammatischen Sprachstörungen" to "Hughlings Jackson, the deepest thinker in neuropathology of the last century," no one attempted to understand his contribution to the subject. Moutier mentions his name along with those of other Englishmen who held more popular views, not knowing how closely Hughlings Jackson's conceptions agreed with those of Moutier's master Pierre Marie. Von Monakow mentions four only of Hughlings Jackson's papers amongst over 3,000 which form the references at the end of his volume on "Die Lokalisation im Grosshirn," and there is nothing in the text to show that he has understood Jackson's point of view or has recognized how completely he stood outside the popular conceptions.

Four reasons may be offered for this extraordinary neglect of Jackson's work. Dr. Hughlings Jackson was a man of such profound personal modesty that he laid little value on the publication of his views, although firmly convinced of their utility in explaining the phenomena of disease. Thus most of his papers are to be found in Journals which are not accessible to foreigners, and many cannot be consulted even by English workers except in the great medical libraries.

Secondly, the style in which they are written makes them peculiarly difficult to read. He was so anxious not to overstate his case that almost every page is peppered with explanatory phrases or footnotes, so that the generalization can scarcely be distinguished from the qualifications. English students, accustomed to the fluent facility of his contemporaries, turned away from the bristling difficulties of Hughlings Jackson's papers.

Thirdly, Jackson derived all his psychological knowledge from Herbert Spencer, and adopted his phraseology almost completely. This has tended to alienate psychologists, blinding them to the truths underlying this somewhat uncouth nomenclature. In this paper I shall keep as closely as possible to Hughlings Jackson's phraseology, and shall not translate it into more modern terms except where obsolete conceptions adopted from others seem to obscure valuable observations.

Finally, the nature of the ideas he propounded was foreign to the current views of the day. He was always accustomed to say that "it generally takes about twenty-five years to become known in medicine," a dictum certainly founded on his personal experience. Each generation of house physicians and clinical clerks at the National Hospital passed out impressed by the beauty of his character and his single-hearted sincerity. Some carried away with them one or more of his broad generalizations to bear fruit in their subsequent work. Thus, for instance, his doctrine that "destructive lesions never cause positive effects, but induce a negative condition which permits positive symptoms to appear," has become one of the hall-marks of English neurology.

But no one assimilated his views on defects of speech and applied them to a series of actual cases of this condition. We failed to appreciate how much closer these conceptions would lead us to the phenomena of aphasia than the glib generalities founded on the anatomical facts of cortical localization.

Although the study of the phenomena of aphasia is one method of elucidating the mechanism of speech, it is by no means the only one. In the last forty years philologists, psychologists, and students of philosophy have steadily progressed away from the conceptions expressed by neurologists, even in their most recent monographs and text-books. Neurology has become frozen stiffly in the grip of pseudo-metaphysical classifications which neither explain the conditions nor correspond to the clinical facts. The dangers of such false classification were recognized by Jackson in 1878: "We must not," he said, "classify on a mixed method of anatomy, physiology and psychology, any more than we should classify plants on a mixed, general, and empirical method as exogens, kitchen herbs, graminaceæ and shrubs" [p. 115].

By an irony of fortune the advance of knowledge, in other than neurological fields, has resulted in the adoption of a position in many ways corresponding closely with that held by Jackson more than five and thirty years ago. This has induced us to republish the most important papers of Hughlings Jackson on Defects of Speech, together with such fragments from other publications as may seem of value at the present time. A bibliography, as complete as possible, has been supplied with a short account of the contents of each paper. Thus we hope to enable others to recognize the foresight of Jackson's views on speech and to assist subsequent workers to make further advance along the lines he has indicated.

CHAPTER I.—HUGHLINGS JACKSON'S METHOD OF STUDYING AFFECTIONS OF SPEECH

Throughout the whole of his work on affections of speech Hughlings Jackson insisted on the necessity of studying and classifying the phenomena before any attempt was made to correlate them with the morphological changes. Speech is a function of mental activity; it is, however, in the last resort ultimately bound up with the integrity of some portion of the brain-substance; but the problem is primarily a psychological one. "We must," he said, "consider our subject empirically and afterwards scientifically. We first, and mainly, [illegible] arrange for convenience of describing the main facts which particular cases supply, and then try to classify the facts in order to show the fundamental relation [illegible] to [illegible] and consider how [illegible] on the psychical side [illegible] effects [illegible] on the physical side are defects of the nervous system" (p. 1[illegible]).

Every worker on the affections of speech is forced to deal with the "facts" of each case, but no one except Jackson has recognized that all the phenomena are primarily psychical and only in the second place susceptible of physiological or anatomical explanation. Even the most modern accounts, such as that given in v. Monakow's monograph, attempt to deal with the clinical facts as if they could be deduced from the physiological activity of morphological centres. A visual centre or an auditory centre demands a visual or auditory form of affections of speech, and efforts are made to classify the psychical phenomena as a direct expression of the activity of such centres. Still greater chaos was introduced by the theory that affections of speech are due to destruction of memory-images, "visual psychic" or "auditory psychic"

Some critics have imagined that Jackson's ideas were built up on a priori reasoning; but anyone who will attempt to examine a case of aphasia according to his conceptions will find himself led to the discovery of phenomena which will elude any other more theoretical method. He will discover in many cases that it is impossible to say that a man can or cannot speak; for he can utter a phrase or a word which he cannot repeat to order. He can write his name and address, but not that of his mother with whom he lives. He may understand all ordinary commands to fetch this or that object, but told to close his eyes, opens his mouth. He may not be able to draw a square or a triangle, but draws an elephant without difficulty. The "golden rule," upon which Dr. Jackson insisted—"Put down what the patient *does* get at and avoid all such terms as amnesia, &c.," had it been habitually followed, would have saved neurologists from many years of wandering in the wilderness.

Jackson's attitude was strictly phenomenal. He never deduced his observations from his hypothesis, but any hypothesis he enunciated sprang, as it were, ready made from some clinical fact. He never experimented or arranged a series of observations to elucidate a definite point. He stood like an observer on a bridge formulating the extent of the flood from matter carried down by the stream. The acuteness and rapidity of the conclusions he drew from some minute indication made his slower-witted companions believe that he had produced the hypothesis from his inner consciousness; whereas, in truth, the view he enunciated was the direct outcome of some phenomenon accurately observed and carried to a strictly logical conclusion.

He did not choose those cases which exhibited the most perfect conditions for experimental observation; he would watch a patient in the throes of delirium tremens as he tossed in the padded room, or listen to the laughter of a man with pseudo-bulbar paralysis, or the recurrent utterance of some partly demented aphasic, and from such observation would spring the luminous exposition of a principle. We did not see how much closer he was to the clinical facts than those who talked glibly of "centres" for psychical processes.

He never failed to recognize that the phenomena of aphasia did not stand alone; they illustrated the same principles and were governed by the same laws as other functions of the nervous system. Above all, he insisted on the importance of remembering that dissolution occurred first in the most highly organized products of neural or mental activity, leaving the more lowly at liberty to express themselves freely in the resulting symptoms.

'The consideration of such facts will help [illegible] phenomena of cases of aphasia [illegible] more [illegible] than [illegible]. To use an expression somewhat loosely, [illegible] the [illegible] of certain voluntary actions in some cases of aphasia will [illegible] of the more automatic—a dissolution affecting [illegible] processes and affecting language [illegible] not so much as [illegible] processes but as they are some of the voluntary [illegible]. We [illegible] consider speech on this wider basis in order that we may be [illegible] to see how speech is part of [illegible], and to [illegible] there is an abrupt and constant separation [illegible] [illegible].

CHAPTER III.—[illegible]

Dr. Jackson's [illegible] clinical lecture in the London [illegible] for 1864 was devoted to the classification [illegible] stress on the importance of separating [illegible] paresis of tongue, lips, or palate from [illegible] pointed out that patients [illegible] but had difficulty in [illegible] freely; [illegible].

Somewhat [illegible] he described cases [illegible] patient could not protrude the tongue on [illegible] but could [illegible] perfectly in a [illegible] his [illegible] [p. 37 and 104].

In 1868 the British Association [illegible] held its annual meeting at Norwich and [illegible] on aphasia, he was followed by Dr. [illegible] record of this paper [illegible] of the speakers; but [illegible] *Cases*, from which we can obtain [illegible] expressed by Dr. Jackson [p. [illegible]].

"Healthy language," he said, "should [illegible] forms, which may [illegible] separated by disease.

"(1) Intellectual, i.e., the power to [illegible]

"(2) Emotional, i.e., the [illegible] to exhibit [illegible]

"Intellectual language [illegible] manifested [illegible] such [illegible] words [illegible] is the power [illegible] which [illegible] in those most [illegible] those [illegible]

Emotional language is preserved throughout not only in its most striking manifestations by variations in tone, but in smiles, &c., and in its more simple manifestation by gesticulation."

Here he strikes the note which sets the key of all his subsequent work. Speech, apart from its articulatory aspect, is double, consisting of intellectual and emotional language, and it is the former that is usually disturbed in consequence of cerebral disease. What, then, is the special nature of these defects of intellectual language? Jackson pointed out that patients with affections of speech fell into two groups.

"Class 1.—Severe cases in which the patient is speechless or nearly so, or in which speech is very much damaged." In the worst of these cases the patient can only utter some one unvarying word or two words or some jargon.[*]

"Class 2.—Cases in which there are plentiful movements, but wrong movements, or plenty of words but mistakes in words." In this class the phenomena show that speech suffers in proportion to the mental complexity of the task the patient is asked to perform. He is not wordless, and knows what he wants to say; but he uses wrong words, has difficulty in consecutive expression both by word of mouth and writing and his speech may be reduced to a jargon. The extent to which all these defects are manifested at any moment depends on the nature of the mental processes necessary to the successful carrying out of the task.

Then he asks "what is the degree of intelligence these patients have?" and expressly guards himself against any implication that language and thought exist separately. "The question," he says, "is not 'How is general mind damaged?' but 'What aspect of mind is damaged?'"

(a) Sign-making is least affected, sometimes seeming to escape altogether.

(b) Writing suffers more or less in nearly every case of defect of speech from disease of the hemisphere, but varies as much as the defect of speech itself does. "Indeed writing, and we may add reading, is the same defect in another form. For in each we have to reproduce motor symbols of the words. Written or printed symbols are 'symbols of symbols.'" The patient who cannot write can usually copy and can often sign his own name when he cannot write anything else.

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[*] Throughout the whole of Dr. Jackson's subsequent papers the term "speechless patient" refers mainly to this class of aphasics. This definition of the use of "speechless" occurs again in the first paper he wrote for BRAIN [p. 110].

and involuntary movements is very striking in some cases of loss of speech; the degree of loss of power to utter words must not be taken as a certain index of this difference. . . . . Anyone who has tried to use the ophthalmoscope in many cases where speech is lost or defective, will find how great the difference is in different cases. . . . . A few months ago a patient came under my care who could only say 'pooh ! pooh !' and an examination of his eyes was almost impracticable. He made [illegible] but he never did what I told him, whether it was to look in a particular way or to keep his eyes still. Instead of opening them, he opened his mouth or screwed up his face, or shut his eyes and could not be got to look in any particular direction, although he seemed to be on the alert to act and was all the time doing something with his hands. . . . Now it may be said that patients do not know what we wish them to do. But (one) patient when told to look at my finger seemed to know what was wanted, for when I was about to give up in despair he took hold of my little finger, as I guessed deprecatingly, and as much as to say he knew what was wanted but could not do it. . . . It will be observed that a speechless patient who cannot put out his tongue when told will sometimes actually put his fingers into his mouth as if to help to get it out; and yet not infrequently when we are tired of urging him he will lick his lips [illegible]. Now, as a rule, the worst of these patients can generally smile, and all such involuntary processes go on well enough. I say generally, for a physician who has seen many cases of defective speech will be obliged to use the word 'generally.'"

Here we have a clear recognition of the variability of response in all such cases of affection of speech. This variability and uncertainty in obtaining the same result throughout a series of observations is one of the characteristics of defective cortical activity and is responsible for much of the difficulty in recording results of a clinical examination in cases of aphasia.

Not only do the results of any series of similar observations vary enormously, so that at one moment the patient seems to possess a faculty of which he is devoid at the next, but all use of such words as "agraphia," "alexia," "amnesia verbalis," and the like is rendered impossible and highly dangerous to the truth of the record by the selective nature of the loss in any one group of tests.

How, for instance, are we to characterize in one word the condition of the man who can write his name, that of his house, the number and the street, the county and kingdom correctly, and yet cannot do the

Moreover he may find no difficulty in copying exactly [illegible] a printed document in cursive handwriting although he is [illegible] to write any of these words spontaneously or to dictation.

(c) Do they know what is said to them? In Class I, [illegible] the patient is speechless except for some recurring utterance [illegible] they usually do understand; but in Class II, [illegible] they have [illegible] disorderly utterance, they often do not quickly understand words said to them. [illegible]

(d) Can they repeat words said to them? In Class I they cannot, whilst in Class II they can with or without blunders.

(e) They cannot read but they often can understand what is [illegible] to them.

He then passes on to show that the general law governing these disturbances of speech is that voluntary power is diminished with retention of the power to carry out the same movement in a more automatic manner. This is illustrated by the example of the speechless man who was unable to protrude his tongue when told to do so, although he could perform this movement perfectly to lick a crumb from his lips.

These speechless patients are not "wordless," for they can swear and even utter [illegible] appropriately on occasion; a man usually speechless may even [illegible] get out an actual proposition. But the words [illegible] his disposal [illegible] they [illegible] comprehension [illegible] also be called upon under [illegible] the movements of the tongue [illegible] cannot be evoked by [illegible]

This aspect of the phenomena in speechless patients had been elaborated in an earlier paper [p. 48].

"In some cases of defect of speech," he says, "the patient seems to have lost much of his power to do anything he is told to do even with those muscles that are not paralysed. Thus a patient will be unable to put out his tongue when we ask him, although he will use it well [illegible] [illegible] There is power in his muscles and in the centres for the co-ordination of muscular groups, but he, the whole man, or the 'will,' cannot set them a-going. Such a patient may do a thing well at one time but not at another. In a few cases the patient cannot do things so simple as moving the hand (the hand not paralysed) when he is told."

[illegible] [p. [illegible]] [illegible] same for his mother with whom he lives? Is he suffering from [illegible] or not? What name is to be applied to the state of a man who cannot read a word of a letter he has received when asked to do so, but who carries out the instructions it contains accurately with regard to time and place? Is he a case of alexia or not? In a similar way the use of the phrase amnesia verbalis lands us in numerous difficulties. Supposing that after the patient has failed to name several common objects he says, when shown the ink—"That's what I [illegible] a china pot to hold ink"—has he amnesia or not? On Jackson's [illegible] the significance of such an answer is clear: the disturbance of voluntary speech destroys the power of making the more selective answer "ink," but [illegible] a more descriptive response. What, again, is the condition of a man who cannot draw a "square" when asked to do so, but when asked to draw a "block of wood" at once draws a perfect square?

CHAPTER IV.—SUPERIOR AND INFERIOR SPEECH.

The idea that speech is a mental process which can be [illegible] by disease into emotional and intellectual expression was developed by Jackson into the differentiation of superior and inferior speech.

"Figuratively speaking, emotion uses propositions in a [illegible] interjectional manner, that is to say reduces them to or towards inferior speech. Emotion, as it were, appropriates and subordinates an intellectual utterance" [cf. p. 163].

The words uttered by an otherwise speechless patient can be divided into "recurring utterances" and "occasional utterances."

Recurring utterances may be:—

(1) Jargon such as "Yabby," "B[illegible], me committimy [illegible] daah." They have no propositional value whatever.

(2) Sometimes the utterance is a word such as "[illegible]" "[illegible]" this is not a word to the patient, for, though a single word [illegible] have a propositional value [illegible] these words have [illegible] for the patient. The [illegible] comes out [illegible] and not appropriately [illegible] person is asked "[illegible] oranges will you have?"

(3) The recurrent [illegible] such as "[illegible]" "Oh, my God!" These [illegible] a proposition [illegible] have in the mouths of speechless [illegible] propositional value [illegible] are at best interjectional.

(4) It is common [illegible] in all his [illegible]

"yes" or "no," or both these words. To speak is to propositionize, and "yes" and "no" are "proposition-words." But they are not always used for assent and dissent, and in so far they are not propositions and are therefore liable to be retained by the speechless patient. He may utter "yes" and "no" without any sort of application, or even the one when he means the other. But although the actual word used may be incorrect, the tone in which it is uttered and the smile or gesture which accompanies it, may correctly express the patient's meaning.

In some cases, however, of loss of speech, there may be a use of "yes" and "no" higher than a mere emotional service; the patient can reply with them. But even in such cases they may not reach the level of normal speech. The patient cannot utter the word in all the ways healthy people can. He may be able to reply "no" to a question requiring dissent, although he cannot say the word when he is told and when he tries to do so.

Occasionally he can not only use "yes" and "no" correctly, but can even repeat them. Here, then, we have propositional speech and voluntary utterance although otherwise these powers have been destroyed.

Hughlings Jackson then deals with the occasional utterances which may occur in the speech of the aphasic patient. These may be —

(1) Utterances which are not speech, such as swearing and ejaculation. The patient may say "Oh, dear," or "Bless my life," but cannot repeat them voluntarily. In some people oaths have become very deeply automatic, almost as much so as [illegible]s; they are so to speak "detonating commas."

(2) Occasional utterances may be [illegible] A speechless patient said, "Whoa, whoa," [illegible] horse; another could say "Goodbye" when [illegible] room, and yet neither patient could repeat th[illegible] ...ately when asked to do so.

(3) He records still higher de[illegible] speechless patients—a woman suddenly asked [illegible] A man who was asked where his tools [illegible] Both of these were of high propositi[illegible] ...e latter; in neither case, however, could the [illegible] ...d voluntarily. They rose to expression under [illegible] emotional stress.

Passing on to a higher leve[illegible] out this mar... patients who can say a word [illegible] cannot say either "batter" or "sea." The [illegible] on account of its gold is yellow—consists of two names, each of which, by conventional contrivances of position, &c. . . . modifies the meaning of the other. All the names in a random succession of words may, it is true, be revived . . . excite perception in us but not perceptions in any relation to one another deserving of the name of thought. The several perceptions so revived do not make a unit. We are told nothing by a mere sequence of names, although our organization is stirred by each of them. Now a proposition is not—that is to say, in its effect on us—a mere sequence.

"When we apprehend a proposition, a relation between two things is given us—is for the moment, indeed, forced upon us by the two conventional tricks which put the two names in the respective positions of subject and predicate. We receive in a twofold manner, not the words only, but the order of the words also" (p. 86).

"Single words are meaningless, and so is any unrelated succession of words. The unit of speech is a proposition. A single word is, or is in effect, a proposition, if other words in relation are implied . . . It is from the use of a word that we gather its propositional value. The words 'yes' and 'no' are propositions, but only when used for assent and dissent; they are used by healthy people interjectionally as well as propositionally. A speechless patient may retain the word 'no,' and yet have only the interjectional or emotional, not the propositional use of it; he utters it in various tones as signs of feeling only. He may have a propositional use of it, but yet a use of it short of that healthy people have, being able to reply 'no,' but not to say 'no,' when told . . . . On the other hand, elaborate oaths, in spite of their propositional structure, are not propositions, for they have not, either in the mind of the utterer or in that of the person to whom they are uttered, any meaning at all; they may be called 'dead propositions.' . . ."

"Loss of speech is, therefore, the loss of power to propositionize. It is not only loss of power to propositionize aloud (to talk), but to propositionize either internally or externally, and it may exist when the patient remains able to utter some few words. We do not mean by using the popular word 'power,' that the speechless man has lost any 'faculty' of speech or propositionizing; he has lost those words which serve in speech, the nervous arrangements for them being destroyed. There is no 'faculty' or 'power' of speech apart from words revived or revivable in propositions, any more than there is a 'faculty' of co-ordination of movements apart from movements represented in particular ways. We must see [illegible] too that [illegible] the [illegible] words [illegible] speech there is a [illegible] use of words [illegible] use the expression that the speechless man has [illegible] he has lost those words which serve in speech [illegible] does not mean entire wordlessness.

"On the psychical side [illegible] loss of words used in speech. The expression [illegible] words' is too indefinite [illegible]. We are [illegible] indeed, [illegible] that the patient has words remaining [illegible] positive condition under the vague expression [illegible] of words.' If we do use such redundant [illegible] thorough [illegible] application of [illegible] we shall say [illegible] the speechless patient has lost the [illegible] in speech; and (2) that he has not lost the [illegible] in other ways. In healthy people [illegible] very [illegible] experiment which [illegible] brutally makes [illegible] demonstrate this; it takes one set of words away [illegible] other set" (p. 113 *et seq.*)

The words removed are those [illegible] in the [illegible] those which remain to the [illegible] used [illegible] extensive damage of speech [illegible] disorders the use of words [illegible] the higher or [illegible] abstract [illegible] propositions [illegible] the patient to fail, not only [illegible] emission [illegible] but [illegible] himself of the [illegible] proposition.

[illegible]

One of the greatest obstacles to [illegible] the diverse use of [illegible] speech [illegible] language [illegible] even been used to [illegible] If we [illegible] by different terms [illegible] comprehend [illegible] difficult [illegible] spoken speech [illegible] written [illegible] may [illegible] been used [illegible] which [illegible] spoken [illegible]

Fortunately Jackson leaves no doubt in which sense he uses the expression "internal speech," and throughout his writings he remains steadfast to the original definition.

After demonstrating that speech is not simply the utterance of words but is essentially the formation of propositions, he points out that there is no difference in this respect between external and internal speech.

"I do not mean," he says [p. 182], "that propositioning occurs only when we speak to tell others what we think, but that it occurs when we, so to say, are telling ourselves what we think. Whether 'gold is yellow' is said aloud or whether it is thought that gold is yellow, there is propositioning, a relation of images to one another in a particular way."

Jackson thought of internal speech as identical in form and nature with uttered speech except that it was not expressed over the vocal organs. He therefore sought for some means of investigating disturbances of internal speech, and believed that writing was invariably defective whenever internal speech was disturbed; writing is the key to the conditions of internal speech.

"The speechless patient not only cannot speak aloud, he cannot propositionize internally. This is shown by the fact that he cannot write. He may copy accurately, and can copy print in written characters, but cannot express himself in writing. He can say nothing to himself, and therefore has nothing to write. For its character as speech it matters nothing whether the proposition be said to oneself or spoken aloud.

"So, then, the speechless patient has lost speech, not only in the popular sense that he cannot speak aloud, but in the fullest sense; he cannot propositionize in any fashion. If this be really so, we must not say that speech is external thought, for there is no essential difference betwixt internal and external speech. We speak not only to tell other people what we think, but to tell ourselves what we think. Speech is a part of thought, a part in which we may or may not externalize. Again it is not well to say that thought is internal speech, for the man who is speechless (the man who has no internal speech) can think. How well or ill he can think we will discuss later. . . . There are two ways in which words serve in thought; speech is but one way, and this, whether it be internal or external, is physiologically a function of the left cerebral hemisphere."

Those who do not limit the definition of speech as we have done, But behind these varieties of formulated speech lie more "subjective" and more automatic processes which precede the revival of words. In the next section we shall try to set out Jackson's views of the nature of these processes and the extent to which they are disturbed in the speech of the aphasic.

§ 8. *The "Proposition" antecedent to Internal and External Speech.*

So far Jackson's nomenclature can lead to no serious misunderstanding. Internal and external speech or their equivalents in other languages have been used with such diverse meanings by various authorities that his clear definition of the use to which he puts these terms cannot lead to confusion. But when he deals with the processes behind internal speech his use of the words "subject" and "object" is liable to lead the reader to reject his views inconsequently. If, however, an attempt is made to discover the principles underlying such phrases as "subject and object proposition," the observer will find an idea which enables him to understand the phenomena of aphasia and to invent tests which bring out the limits and nature of the loss of speech in any particular case.

"In all voluntary operations," he says, "there is preconception. The operation is nascently done before it is actually done; there is a 'dream' of an operation as formerly doing before the operation; there is dual action. Before I put out my arm voluntarily I must have a 'dream' of the hand as already put out. So, too, *before* I can *think* of now putting it out I must have a like 'dream,' for the difference betwixt thinking of now doing and actually doing is, like the difference between internal and external speech, only one of degree" [p. 168].

Jackson saw clearly that the process of forming a proposition to be expressed by internal or external speech was a double one. First the mind must be aroused in propositional form and then the words must be fitted to the proposition. This fitting of words to the proposition is internal or external speech, according as it finds utterance to oneself or to the outside world; the process which precedes this is what he called the "subject-proposition."

"If we coin the term *verbalizing* to include the whole process of which speech is only the end or second half, we may say that there are in it two propositions: the subject-proposition followed by the object-at any rate by internal, and in some, especially the less educated, by external speech. But internal and external speech are not necessary to perfect logical thought, whilst the process called by Jackson "subject-proposition" forms an essential portion of the act of thinking.

Behind this "subject-proposition" stands that mental process included by Jackson under the name of "image" or "symbol," and this forms the subject of the next section of the chapter devoted to his views on speaking and thinking.

§ 4.—"*Imperception*" *and the Condition of the* "*Images*" *in a Speechless Patient.*

No aspect of this work exhibits Jackson's peculiar insight more clearly than his treatment of the mental processes which stand behind the birth of a propositional statement. For at a time when everyone assumed that the destruction of auditory and visual word images was a sufficient explanation of the phenomena of aphasia, he saw that in the speechless man these images might be intact.

He divided cases of speech defect of central origin into two groups, those where images were intact and those in which they were defective.[1] To the latter he gave the name "imperception" and pointed out that this state corresponded on the afferent side to aphasia in the word series. Many years before the invention of the term "agnosia" Jackson clearly described this condition under the term "imperception" and illustrated his description with clinical instances.

In many cases met with in practice, aphasia and imperception are combined; but the great merit of Jackson's observations is the clearness with which he saw the necessity of separating these two factors in any particular example. At that time everyone believed that the destruction of "images" was a simple explanation of aphasia. Jackson showed that there were affections of speech in which "images" might be defective, but, on the other hand, they were certainly not abolished in a large number of cases of aphasia.

"We must then briefly consider," he says [p. 121 *et seq.*], "the patient's condition in regard to the images symbolized by words. For although we artificially separate speech and perception, words and images co-operate intimately in most mentation. Moreover there is a images, although with more difficulty, they, besides lacking their accustomed stimulus, being less organized. He can copy writing and can copy print into writing. When he comes print into writing, obviously he derives the images of letters from his own mind. He does not write in the sense of expressing himself, because there are no words reproduced in speech to express. That series of artificial images which make up the signature of one's own name has become almost as fully organized as many ordinary images; hence in many cases the speechless man who can write nothing else without copy can sign his own name" [p. 133].

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[1] Cases of so-called "Mind-Blindness," where this power is lost, are due to extensive "imperception" and in so far [illegible] this discussion.

Jackson recognized that in the speechless person we possessed a means of analysis of the processes behind the formation of speech. Images, whether direct or symbolic, together with those unconscious mental processes on which they may depend, remain intact in the speechless patient. He cannot speak, he cannot write, he cannot read, not because he has lost "images" or "memories" of words, but because he cannot propositionize. He has lost the use of words in speech.

Those cases where images are affected suffer from a distinct and definite defect which he calls "imperception." Speechlessness may exist without imperception, and it is in the [illegible] cases only that we can obtain a clear analysis of the processes which make up speech.

§ 3.—The Mind of the Aphasic.

Jackson's method of treating the phenomena of loss of speech entirely from the psychical aspect robs the question as to the condition of intelligence in the aphasic patient of half its significance. In his Norwich address he laid down that the question was not "How is general mind damaged?" but "What aspect of mind is damaged?" He then proceeded to indicate one by one the changes we have considered in previous sections of this chapter.

But when a man has lost the power of propositionizing and cannot speak or write, when both internal and external speech have been destroyed and the process which stands between them and his perceptions is also disturbed, it is obvious, as Jackson points out, that he must be "lame in thinking." The reception of complex and novel propositions demands internal speech for that formulation which must precede their committal to memory. Thus the existence of "inferior speech" is associated with distinctly "inferior comprehension."

Suppose, however, "imperception" is added to the defect [illegible] the formation of images, arbitrary symbols and those [illegible] processes which precede their development will be [illegible]. The "general intelligence" will then appear to suffer greatly, for [illegible] [illegible] be attacked, not only on its emissive side, but also on its receptive side.

Thus Jackson said that the question was not one of loss of [illegible] intelligence," but the disturbance of certain activities [illegible] associated with those lesions of the brain which could affect speech.

CHAPTER VI.—SPEECH AND CEREBRAL LOCALISATION.

It is not to be wondered at that a man who held these views [illegible] aphasia and analysed the phenomena exhibited by the patient [illegible] in this manner remained incomprehensible to his contemporaries [illegible] even to the younger generation. The air was thick with [illegible] [illegible] of centres; all knew there were "auditory" [illegible] and "motor-word centres," "centres" for writing and [illegible] even for ideation.

"It is well to insist again," he says in 1878, "that the speech [illegible] psychical states words have of course anatomical substrata [illegible] as all other psychical states have. We must [illegible] between words and their physical bases as we must between other [illegible] physical bases; a psychical state is always accompanied by a physical state, but, nevertheless, the two things have distinct natures. Hence we must not say that the 'memory of words' is a function of [illegible] of the nervous system, for function is a physiological term.

"A method which is founded on classification which is partly anatomical or physiological and partly psychological [illegible] confuses [illegible] issues. These mixed classifications lead to the use of such expressions as that an idea of a word produces an articulatory movement; [illegible] a psychical state, an 'idea of a word' (or simply 'a word') [illegible] produce an articulatory movement, a physical state" (p. 106).

"The anatomical substratum of a word is a nervous arrangement [illegible] [illegible] special movements of the articulatory organs. [illegible] [illegible] idea of a word it suffices that the nervous process for those movements [illegible] is not necessary that it be energized so strongly that currents [illegible] articulatory muscles. Here it is that, from our negative [illegible] [illegible] kind or a fragment of any sort of innervation [illegible] ideas [illegible] point we are here concerned with" (p. [illegible]).

"The notion that movements or cerebral [illegible] nervous arrangements [illegible] themselves serve in mentation is grotesque. The statement is that a word, a psychical thing, is concomitant with discharges of certain cerebral arrangements representing one or more articulatory movements—and that words serve in mentation" [p. 177].

From his first paper in 1864, throughout the whole series he emphasizes the psychical nature of the phenomena of loss of speech. These changes in mental activity are associated with material destruction of the cerebrum, but it is fallacious to attempt to localize the various activities into which speech may be analysed by introspection, to disease in various portions of the cortex:

"Whilst I believe" he says [p. 81] "that the hinder part of the left third frontal convolution is the part most often damaged, I do not localize speech in any such small part of the brain. To locate the damage which destroys speech and to localize speech are two different things. The damage is in my experience always in the region of the corpus striatum."

He also pointed out that the nearer the disease lay to the corpus striatum the more likely is defect of articulation to be the striking feature of the case: whilst the farther off the disease lies from this part, the more likely will the main characteristic be mistakes in words.

In his Norwich address in 1868, he pointed out that affections of speech of cerebral origin could be divided into two classes: Class I, in which the patient is speechless, or nearly so, or in which speech is very much damaged; Class II, in which there are plentiful words but mistakes in words. In the first class the disease tends to lie in the neighbourhood of the corpus striatum, whilst in the second class it reaches farther back or deeper in the brain.

Even in 1866 he indicated the fundamental error in Broca's localization of the "faculty of speech" and the "memory of words." In an extract from the *Bulletins de la Société Anatomique* for July, [illegible], Broca says, "Pour expliquer comment un aphémique comprend le langage parlé sans pouvoir cependant répéter les mots qu'il vient d'entendre, on pourrait dire qu'il a perdu, non la mémoire des mots, mais la mémoire des moyens de coordination que l'on emploie pour articuler les mots." Jackson takes up this definition [p. 51] and points out that the use of the word memory in the sense of its being a distinct faculty is likely to lead to confusion. The speechless man understands because words remain to him in so far as they are excited from without; he cannot speak because he has lost the service of words in propositions. The words he retains in non-propositional forms may be perfectly words or jargon. Class 2 comprises those who have "plentiful words," but habitually use them wrongly.

(2) The loss of power to carry out an order depends on the complexity of the task. The more abstract the conception the more likely is the patient to fail in executing it, although he may succeed when it is put before him in a simpler and more descriptive form. Thus the patient who could not find the word "kitten" called it a "little fur-child," and one who could not draw a square when asked to do so drew a perfect square when told to draw a "block of wood." Thus it is most important when asking an aphasic patient to carry out some order to present it to him if possible in several forms, noting accurately his response in each case; because a man cannot write the alphabet we must not assume he cannot write a letter.

(3) The higher and voluntary aspects of speech tend to suffer more than the lower or more automatic. The least voluntary speech is that which is emitted under the effect of emotion, such as exclamations, oaths, and words, such as "good-bye," rising to utterance under the impulse of a moment. In many cases "yes" and "no" can be used appropriately as propositions, and even words or phrases of true propositional value may spring to the lips of the aphasic patient. But in such cases he is usually unable to repeat at will the phrase he has just used under an appropriate impulse. The "speechless man is not wordless," and the apparent inconsequence of observations on persons with aphasia is to a great extent removed by an analysis of the conceptual value of the words and phrases which are actually uttered.

(4) Writing is affected, not as a separate "faculty," but as a part of the failure to propositionize in words. There is no such condition as pure "agraphia." A man who cannot write spontaneously may be able to copy printed matter in perfect handwriting. The "faculty" of relating handwriting to print is intact; he cannot write voluntarily because he has lost the use of written words in propositions. Hence he can usually write his name and address, because in most of us this has reached with time more nearly the level of an automatic act.

(5) Patients with such affections of speech may not be able to read aloud or to themselves when asked to do so; but they can understand what is read to them and may even obey written commands although unable to reproduce them in words. This is not due to some loss of function called "alexia," but to an inability to reproduce a proposition which, on the other hand, they may be capable of receiving accurately.

(6) "Imperception" (agnosia) is on the receptive side what aphasia

[illegible] in the word series." In many cases the two con[illegible] combined, but they [illegible] exist separately and [illegible] without [illegible]ception, images remain [illegible] be able to point to colours and objects when they are [illegible] continues able to play cards or dominoes, he [illegible] although he cannot read the words written, he knows [illegible] page by the different endings of the [illegible] the right [illegible]

Thus affections of speech [illegible] (a) on the [illegible] inability to form or to express a proposition in word[illegible] [illegible] by nature of those [illegible] processes [illegible] perceptual recognition.

(7) External and internal speech [illegible] [illegible] leads to the [illegible] of articulated words, [illegible] internal speech can be discovered [illegible]

(8) Behind external and internal speech stands [illegible] which when verbalized can be expressed in speech [illegible]

This [illegible] is necessary for [illegible] all thinking. When this aspect of [illegible] is affected [illegible] cannot retain a sequence of abstract propositions [illegible] formulate them at will to himself [illegible] in thinking [illegible]

(9) If, however, [illegible]ception (agnosia) is added [illegible] [illegible] the patient [illegible] not only suffer from [illegible] show signs of [illegible] comprehension.

[illegible] the majority of cases of affections of speech [illegible] [illegible] This extremely important [illegible] aphasia has been entirely [illegible] every hypothesis propounded in the last [illegible] defect in "auditory" or [illegible] word [illegible]

REPRINT OF SOME OF DR. HUGHLINGS JACKSON'S PAPERS ON AFFECTIONS OF SPEECH.

For permission to reprint these papers we are indebted to the kindness of the Editor of the "British Medical Journal," the Editor of "The Lancet," the Editor of "The Medical Press and Circular," and Messrs. Churchill, the proprietors of the now extinct "Medical Times and Gazette."

LOSS OF SPEECH. ITS ASSOCIATION WITH VALVULAR DISEASE OF THE HEART AND WITH HEMIPLEGIA ON THE RIGHT SIDE.—DEFECTS OF SMELL.—DEFECTS OF SPEECH IN CHOREA.—ARTERIAL LESIONS IN EPILEPSY.

[*Clinical Lectures and Reports London Hospital*, 1864, vol. i. p. 388.]

I BRING forward, in this paper, thirty-four cases of hemiplegia, in all of which loss of speech, in a greater or less degree, was present. In thirty-one the hemiplegia was on the right side, in three on the left. In two additional cases there was no hemiplegia at all.

The chief physiological results of the following observations I arrived at independently, but on every point of importance I have been anticipated by M. Broca. Therefore, what I have to do is simply to bring forward my work as evidence bearing on his views. Besides, M. Broca has studied the subject from another point of view, and has arrived at more precise results than I can possibly deduce from observations which are clinical only. M. Broca believes that disease of the left side of the brain only, produces loss of language; and, moreover, he locates the faculty of articulate language in a very limited part of that hemisphere. My observations tend to support the first hypothesis, and, in a general way, the second. The convolution of articulate language of Broca is but one of many convolutions supplied by the left middle cerebral artery, which vessel I believe to have been plugged in most of the following cases of "Loss of Speech with Hemiplegia on the Right Side." Therefore, my observations must necessarily be indefinite evidence as to the exact seat of the faculty of language, or of articulate language, but fairly definite as to the side of the brain in which these faculties reside.

Then as to the clinical part of the paper. [illegible] arteries is now an old and established subject, and [illegible] embolism to valvular disease we are indebted to the researches of Dr. Kirkes. Here, again, I have no novelty of any kind to bring forward, but I think the concurrence of hemiplegia [illegible] and valvular disease of the heart has not been generally noticed. I wish to draw attention to the fact that in Dr. Kirkes's first paper [illegible] first cases of hemiplegia from embolism of the middle cerebral [illegible] ever recorded) it is mentioned that there was loss of speech [illegible] hemiplegia.

Again, the various kinds of defects included [illegible] "Loss or Defect of Speech," and which will be discussed [illegible] have long been described; as, for instance, in M. Broca's [illegible] Siège de la Faculté du Langage articulé [illegible] Dr. Brown Séquard, my late colleague at the Hospital for Epilepsy and Paralysis, for first teaching me to distinguish from one another [illegible] several cases—defects of language, speech, articulation, &c.

I have premised the above as to priority, simply to have done with it, and in order that I may give with more freedom, [illegible] way, the steps by which I arrived at the results announced [illegible] I remark then, once for all, that although I have in a great part [illegible] subject worked quite independently, I lay no claim to priority [illegible] point of importance.

I wish also to draw attention prominently to the [illegible] personal experience of these cases is clinical only. [illegible] chronic ones, and I have not had a single *post-mortem* [illegible] hemiplegia or any case with loss of speech and [illegible] then, of necessity, keep almost entirely to the clinical aspect [illegible] little or nothing about the exact convolution of the [illegible] which produces loss or defect of speech. In the [illegible] M. Broca's paper, more definite information will be [illegible] subject. He gives the [illegible] It is obvious [illegible] in the physiological study of the convolutions of the brain, and [illegible] is the only trustworthy means of getting precise information. [illegible] is equally obvious that we can deal of our own work [illegible] beforehand if our pathological facts are to have any [illegible] physiological evidence.

Besides the physiological value and clinical importance, the [illegible] great pathological interest. [illegible] highly important. But here, again, I am [illegible] as possible from the clinical study of the cases. My object now is briefly this, to try to show under what circumstances, and in association with what other symptoms, loss or defect of speech occurs, rather than to discuss the mental bearings of the symptom itself. To do the latter would require a vast amount of time and space, and would, besides, draw me into a subject to which, I confess, I have not yet paid the attention it deserves. I am glad to be able to say that M. Broca is now preparing a work which will include all that he has done in the matter. This renders it still less necessary for me to do more now than define the term I must use for my present purpose—to circumscribe the symptom rather than to show its psychological relationship to other definite mental faculties or to mind generally. In what follows, then, little more will be attempted than to distinguish the mental symptoms of the cases, which I include under the word "speech," from other defects in talking which bear a superficial resemblance to them, and with which they might easily be confounded. I wish, in short, to speak of the cases as they appear to medical practitioners.

In the first place, let me say, once for all, that I do not mean mere difficulty in utterance from more or less paralysis of the lips,[1] tongue and palate, but a mental defect, more or less loss of language, varying from the most general to the most special manifestation of that faculty. Under the word "speech," then, I include all grades and varieties of expression of ideas, chiefly by words, as distinguished from the mere utterance of words by the lips, tongue and palate.

Talking, in the conventional sense of the word "talk," implies three inseparable but distinct things—voice, articulation and speech. The first is for sound, as in singing; the second for utterance of words; and the third for the expression of ideas. Voice by the larynx, articulation by the lips, tongue and palate, speech by the brain. Therefore, in disease of the nervous system, we have to attend to three things when we hear our patient talk: (1) The sound of his voice; (2) the articulation of his words; and (3) the sense of what he says, and how he says it. The third or mental defect is the one chiefly involved in the following cases.

M. Broca speaks of two kinds of loss of language; first, a loss of the general faculty of language; and second, a loss of the faculty of articulate language.

The first implies a total loss of power to communicate by words, by writing or by signs, or in short in any way whatever. If we ask a patient who has lost altogether the general faculty of language a question requiring merely assent or dissent, he cannot reply even by a sign. He may nod, but not at the right time, just as when he [illegible] but the word "no"; he will say this in return to any question [illegible] will be able to answer none.

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[1] I mention the lips, tongue and palate together, as we often find them paralysed at the same time.

The second is a loss of but one special form of language, viz., that by words. This—the loss of the faculty of articulate language without any other mental defect—is what M. Broca calls aphemia. [illegible] born deaf has no *articulate* language, but his general faculty [illegible] well developed; and he may converse, not only rapidly, but with precision, by his fingers (deaf and dumb alphabet).

Aphemic patients, then, have lost but one special form of language, and, like the deaf mute, they can still express themselves by writing, signs, &c. Now M. Broca finds that in such cases but one special part of the brain is invariably damaged, viz., *the posterior part of the third left frontal convolution.* This is the "convolution of articulate language."

Since M. Broca first drew attention to this subject, there has been recorded but one exception to his views as proved by an autopsy. In this unique case the disease was very near, but did not involve, the third left frontal convolution. [illegible] moreover, so sudden [illegible] that one may well ask if there [illegible] not [illegible] minute changes in the convolution of articulate language which escaped observation. It is well known to all who have read Lockhart Clarke's papers, that parts of the nervous system which look absolutely healthy to ordinary examination are often found to be much diseased when very carefully examined.

The lesion occupied the (left) parietal lobe, and was prolonged across the fissure of Rolando as far as the transverse (left) frontal convolution, which was diseased just at the point where it is joined by the *third left frontal convolution*. [illegible] this last there was no lesion of any importance. It will be [illegible] found that in [illegible] otherwise [illegible] case, the disease [illegible] the left side of the brain.

Now, the word "aphemia," as M. Broca uses it, will not apply to the defect in most of the cases I am about to relate. In these it is clear, as Dr. Brown-Séquard pointed out, that the patient has lost altogether the general faculty of language, and not merely the [illegible] patient's manifestation of that faculty by articulate words. Inability to talk was, it is true, the most marked feature in the case, but it was not more characteristic than the inability to express ideas, by writing or by signs. It will no doubt seem a sweeping statement, but it is a fact that the patient never made me a reply in any way all the time he was in the hospital.

Yet it must be kept in mind that, as I have already said, M. Broca points out but one *limited part* (the posterior third of the third left frontal convolution) as the seat of the faculty of articulate language—as the part always damaged in aphemia—whereas, in at least some of the cases (of embolism of the left middle cerebral artery), I relate there will be far wider damage of the hemisphere. Not only will the "convolution of articulate language" be diseased, but many other convolutions as well. Therefore, although there is aphemia, yet there is something more: not only a loss of power to express ideas by words, but so much mental defect besides, that the patient cannot express himself in any other supplementary manner; and, in some cases, perhaps, cannot find ideas to express.

I must, therefore, keep to the word "speech," and use it as a general term for all shades of intellectual expression, from the most general to the most particular. This is, I admit, a vague way of using the word, but it would do far more harm to use a more definite term, when precision is in reality impossible; and I must beg the reader to remember that it is not used in a dogmatic sense. It is used for the sake of arranging clinical facts, and not as a permanent definition. But I have, as far as I could, described the defect in recording each case, and it will be found that in many of them there is very much more than mere inability to utter words.

The reader will readily understand that it would not be very difficult to compile an essay on language or on speech. But I submit that it would be far from easy to translate into the special language of psychology the variable and undefined effects in the following cases. In a subject so wide and vague as language, it would be simple work to pile up ingenious theories, but to find a method to arrange the varying facts in many actual cases is quite a different thing.

Next, at the risk of being tedious, I enlarge on what I do *not* mean by loss of speech. I speak then of the two remaining factors of "talking"—voice and articulation—in order more effectually to exclude them.

It will scarcely be necessary to say much about voice. Aphonia speech, due to want of power in the lips and [illegible] the [illegible] syllables of patients who are partially [illegible] are recovering from hemiplegia. [illegible] When a hemiplegic [illegible] talks [illegible] although able to move the [illegible] but he was [illegible] I believe it certain that the defect [illegible] to [illegible] of the [illegible], near the [illegible], and [illegible] of the [illegible] language in Brain.

[illegible] paralysis of the [illegible] does not [illegible] the patient unable to talk. Neither does [illegible] paralysis of the tongue [illegible] is totally paralysed [illegible] it is just possible that [illegible] might be [illegible] would [illegible] be the [illegible] not talk [illegible] would be quite [illegible] the tongue could not get out the words [illegible] speech altogether [illegible] not [illegible] he could, could not [illegible] thought [illegible] language.

[illegible] speech [illegible] evidence [illegible] between [illegible] from [illegible] the general [illegible] from abolition of power in the tongue, lips and [illegible] the patient have enough power [illegible] or try to get them [illegible] tongue [illegible] however [illegible] penmanship [illegible] gibberish, [illegible] would [illegible] defect [illegible] would show [illegible] expression generally [illegible] the [illegible] by the muscles of the [illegible]

Dr. [illegible] and Dr. [illegible] a remarkable [illegible] the [illegible] could not, for more than a [illegible] to write [illegible] could [illegible] the [illegible] the [illegible] this [illegible] A [illegible] had [illegible] is always due either directly to disease in the larynx, or [illegible] to disease of the nerves to its muscles. There is not [illegible] any medical man would take aphonia for failure of speech [illegible] even if there were other symptoms of brain disease. Of course, aphonia may be a symptom of disease of the nervous system [illegible] never due to an injury of that part of the nervous system [illegible] sphere which is the seat of [illegible]. But it must be kept in mind [illegible] that patients frequently do not make the distinction [illegible] with chorea limited to the *left* side, told me, [illegible] other [illegible] to a question, that she sometimes lost "speech." [illegible] she merely lost her "voice," remaining able to whisper. [illegible] at Mr J. N. Radcliffe's notes of her case, I found that [illegible] is described as "aphonia." Several of the patients [illegible] heard one patient sing many a time after she had lost speech [illegible] recovered; but she always made mistakes in the words [illegible], omitting some, slurring over others, and changing the syllables [illegible]. I need scarcely say that a patient who has aphasia from disease [illegible] might have, at the same time, aphonia from disease of the [illegible] or its nerves. I have, however, not yet seen this coincidence.

"Articulation" is the third element of talking. [illegible] of voice. A person may articulate well, of course in a whisper, even when his throat is cut. I saw a few months ago in the [illegible] Hospital, under the care of my colleague Mr Hutchinson, a patient who could whisper, so as to be understood across the ward, when at the same time his vocal cords could be seen through a wound in [illegible].

Now the muscles of the lips, tongue and palate [illegible] two chief actions, articulation and swallowing. When [illegible] badly from defective power in these parts, it will, I think, be found that they act badly in swallowing too. Whereas [illegible] talk badly from any degree of loss of speech [illegible] "speech" in this paper, can swallow quite well. The [illegible] respect between a patient who cannot speak at all [illegible] hemisphere, and one who cannot utter a word [illegible]

[illegible] voice is the [illegible] of the emotion. It is altered by [illegible] states of feeling, common to men, rather than more special to the [illegible] [illegible] languages, an eloquence [illegible] fusion of passion [illegible] to the speech of the [illegible] [illegible] the dearest talk and language common to all [illegible] speech is the token of Nature [illegible]

I here mean by defective articulation, a defect [illegible] of power in the tongue and palate, the cords, and not the defect I shall [illegible] in connection with the talking of [illegible] recovering from aphasia, which [illegible] for the [illegible] of syllables.

muscles concerned in articulation is very [illegible] swallows readily, the latter [illegible] that there is no exact [illegible] between the perfection of the [illegible] This is worthy of note [illegible] I am convinced that [illegible] defects of articulate [illegible] disease of the hemisphere, [illegible] of the lips, tongue [illegible] on the right (and indeed [illegible]

As I have said there is hemiplegia [illegible] general sense, in [illegible] a little [illegible] of the face, and a little of the tongue [illegible] just [illegible] he said that [illegible] whose case I describe—for instance [illegible] quite [illegible] cases that [illegible] tongue greater than [illegible] nothing whatever to do [illegible] can only [illegible] a "chair" or "potato," especially [illegible] patient's know [illegible] mistakes in [illegible] lation of words [illegible] the lips and tongue [illegible] a gentleman [illegible]

[illegible] the defect in articulation [illegible] few days. The only thing with which [illegible] is the talk of aphasia [illegible]

[illegible]

which there has been no hemiplegia; and of these three had had epileptiform convulsions on the right side; in the fourth the convulsions were said to have affected both sides.

Now, the strangeness of this association—the loss of a purely mental faculty with paralysis of the limbs on one side—made me think frequently on the subject, and seek for some explanation of the concurrence of the two symptoms. For here are (if I may use the expression) two incoherent symptoms—yet each of them quite definite—coming on suddenly together. Then, apart from this, I often wondered how it was that there were no well-defined mental symptoms in cases of embolism of the middle cerebral artery. This vessel supplies the corpus striatum, the highest part of the motor tract, and hence the hemiplegia was easily accounted for: but it also supplies a vast tract of convolutions, and, therefore, one would, a priori, expect to find decided symptoms of mental failure. (This difficulty still remains in respect to cases of plugging of the right middle cerebral artery.) But it was not very difficult to put this on one side, as there are many records of cases of extensive disease of one hemisphere with no symptoms of any kind, at least none that could be detected. It is generally held that the brain is a double organ, and that if one hemisphere be damaged the other will answer the ordinary intellectual purposes of life. The recent researches as to the seat of the faculty of language render this at least doubtful.

Of course I examined the heart in cases of hemiplegia, almost as a matter of routine, just as I did the urine, and in some cases I found valvular disease, and in some I did not. It occurred to me, however, that in cases of hemiplegia with loss of speech, there was nearly always valvular disease, and I then imagined that plugging of the middle cerebral artery might account, not only for the hemiplegia, but, by producing softening of part of the hemisphere, for the loss of speech too. In the cases I first got together for a paper, read at the Hunterian Society, the frequent coincidence of valvular disease was very striking. Of nine cases there was valvular disease in six; but since I have collected many more cases the proportion has been very much less.

In each of the first nine cases the hemiplegia was on the *right* side, indicating, of course, disease on the *left* side of the brain, and, as I further inferred from the heart disease, plugging of the middle cerebral on the *left* side. This was a prominent feature, but I then thought that it might be only a coincidence. When I had reached twenty cases it became more strange; and now that I have seen one exception I cannot but feel that it is very remarkable.

Of course, hemiplegia is not a necessary accompaniment of loss of speech. These two symptoms frequently occur together simply because the part of the hemisphere in which is the seat of the faculty of language, or of articulate language, is near the upper part of the motor tract—the corpus striatum; so that, from mere relation of contiguity, they often suffer together. What damages one of these parts is very likely, at the same time, to damage the other, or afterwards to extend to it; or, as I suppose happens in cases of embolism, because both of these parts are supplied by branches from one arterial trunk—the middle cerebral—so that when the vessel is plugged both lose their supply of blood at the same time. But just as hemiplegia (on the right or left side) occurs without loss of speech, from the lesion being confined to the motor tract and not extending to the convolution of language, so the converse, loss of speech occurs without hemiplegia, the lesion then being confined to the convolution, or convolution of language, and not extending to the motor tract. M. Broca has seen three cases of traumatic and two of spontaneous aphemia without any paralysis. Then, as I have already remarked, we have loss of speech with convulsions on the right side; but to this point I shall refer later.

Yet the frequent connexion of loss of speech with hemiplegia is striking. A reference to Romberg, Abercrombie, Todd, Forbes Winslow, &c., will show this. Dr. Watson relates one case; Mr. Dunn has recorded a very interesting one (*Lancet,* October 22, and November 2, 1850). In Dr. Watson's, and in Mr. Dunn's case the hemiplegia was on the right side.

As I have said, most of my cases are chronic, and I have had no decisive autopsy on any one of them. When the middle cerebral artery is plugged the patient is often killed by this accident. Sometimes he becomes hemiplegic, and, as I believe, in others, subject to unilateral epileptiform seizures. Now, of course, I cannot be absolutely certain that embolism was the cause of the symptom in the following cases, not even in the cases of valvular disease, but I submit that it is almost certain that it was in most of them.

Syphilis certainly does produce disease of arteries, and thus one of the vessels of the brain may have become plugged from coagulation of blood on its roughened surface; if of the middle cerebral artery, hemiplegia would result. The common way, however, in which syphilis produces hemiplegia is by inducing disease on the surface of one hemisphere. There must, of course, be some secondary disease in the motor tract as well, but the obvious *post-mortem* change is disease in the pia mater on the surface of the hemisphere.

* * * *

[Here follows a description of 38 cases of speech defect associated with hemiplegia. The clinical records are short, and do not add materially to our present knowledge. They show conclusively that embolism and syphilitic vascular disease affecting the left hemisphere may cause loss of speech. Throughout these reports of cases interjected comments show how many of the author's views on disorders of speech were already formed, to be expressed more fully later. The longest and most important of these comments, an addendum to Case 32, on the nature of "Swearing," is here reprinted as the close of the paper.]

* * * *

Swearing is, strictly speaking, not a part of language. It is a habit which has grown up from the impulse to add the force of passing emotions to the expression of ideas. It belongs to the same general category as loudness of tone and violence of gesticulation. The distinction of these from language as an intellectual act, may be best illustrated by the remark Dr. Johnson once made to a boisterous antagonist, "Sir, you raise your voice where you should enforce your argument." Although oaths differ from mere alterations of tone, in that they consist of *articulate words*, they are generally used in talking, not to express ideas, but to make up by vigour in delivery what is wanting in precision of expression. They may, indeed, be considered as phrases which emotion has filched from the intellect, to express itself in more definite terms than it could do by mere violence of tone or manner. For oaths had once an intellectual meaning; they expressed ideas, and were uttered with a definite intention. Curses have, in fact, formed an element in all religious services; but nowadays intentional cursing is obsolete; it has degenerated to meaningless swearing, which, like cadence and gesticulation, is but a kind of commentary of the emotions on the propositions of the intellect. Vulgar people insert an oath "at the proper intervals of their speech," as a sort of detonating comma, and thus they render forcible statements which might otherwise strike their hearers as commonplace.

Upon this broad principle, too, that ejaculatory expressions are prompted by the emotions and not by the will, we may explain the expression, "—bless my life!" surprised out of the patient, Mrs. H., by a sudden and unusual excitement. It was the more remarkable here, as these were the only real words, except "yes" and "no," the patient ever uttered. It is clear that such a phrase is used with the same levity as an oath, and without any intention to express the idea which the words really imply. It is not used with the intention of *saying* anything. In the instance referred to the expression was excited just in the same way as an oath in Case 1. The only word that that patient ever uttered during his stay in the hospital, except "yes," which he could utter at any time, was an oath he once brought out when he was irritated. The difference of sex will probably account for the difference in the form of the ejaculation in the two cases. Even words so simple as "Oh dear! oh dear!" when used in anger or vexation, belong to the same category, for then these words, like an oath, are used rather as the clothing of the feeling than for the expression of an idea. I submit, then, that oaths and similar interjectional expressions, are not parts of speech in the broad sense in which the words that form them are, when used to convey intellectual propositions.

Perhaps the explanation of the way in which these phrases are, so to speak manufactured is that they are reflex. Indeed, although very rudimentary mental processes, they seem to me to illustrate the doctrines of Professor Laycock on the reflex function of the brain, and especially illustrate his views. It is quite obvious that they are not voluntary, as the patients cannot repeat the phrases. The will cannot act, but, somehow, an emotion, e.g., anger, gets the words passed through the convolution of language. Just as a paralysed foot will jump up when the sole is tickled, so these words start out when the mind is excited. Such ejaculations as I have mentioned would have become easy of elaboration by long habit, and would require but slight stimulus for perfect execution.

The following instance seems to belong to the same category, although the action was a less fine one, and the stimulus more coarse and definite.

Some years ago, I was on an omnibus, and we were kept some time, as one of the horses would not start. Various plans were tried to overcome its stupidity, but without success. At last the driver directed the conductor to shut the door violently (this is a usual signal for starting). To my great surprise, the horse went on at once. The horse would not exercise his will to move; the patients could not exercise theirs to talk. But the movement required was surprised out of the unwilling horse by a noise to which he was accustomed to hear in starting. So the anger or surprise being the stimulus under which they had been uttered when the will was sound, gave rise to ejaculations in the patients whose voluntary power to talk was lost.

I have a patient, the subject of chorea, who, for several years, has been in the habit of saying, quite involuntarily, the word "bloody." A few years ago, he was frightened by a man shouting the word after him. The fright produced chorea, and, if I may use such a term, chorea of his mind, too; as, for three days, he said the word "bloody," and little else; and now he ejaculates it occasionally. The mental process for saying that word is as little under his control as a few of the muscles of his face are, for the twitching of which he is now attending at the London Hospital.

CLINICAL REMARKS ON EMOTIONAL AND INTELLECTUAL LANGUAGE IN SOME CASES OF DISEASE OF THE NERVOUS SYSTEM.

[*The Lancet*, February 17, 1866, vol. i, p. 174.]

Development of the Difference between Emotional and Intellectual Expression.

At a recent visit to the London Hospital, Dr. Hughlings Jackson pointed out to us the case of a man who had lost speech, and who was paralysed on the right side. We have already given a *résumé* of Dr. Jackson's views on cases of this kind (*Lancet*, November 26, 1864); and now we speak on different aspects of the same subject as illustrated by cases under his care at the London Hospital and at the National Hospital for Epilepsy and Paralysis.

The patient we saw could only say the word "dick," and this word he uttered whenever we asked him a question. We were told that when the man was vexed by the other patients in his ward he would swear. He generally used the common explosive sound so much in favour with English swearers. He could not, however, say the word when required to do so, even whilst it was well kept before his mind by frequent repetition. He seemed to make efforts to say it, but the word "dick" always came out instead. The oath was only uttered under the influence of emotion, and could never be repeated at will. Oaths—that is, as they are vulgarly used—are little better than more or less highly compound interjections. It is not safe, then, to conclude that a patient who has lost speech is regaining power of language because he begins to swear when he is excited. By such words no part of a proposition can be conveyed; that is, they add nothing to precision of expression in delivering an idea, although they may help the speaker to show states of feeling, and thus to excite sympathy. Where no proposition is conveyed, there is no intellectual language. It is true that some oaths, considering the mere arrangement of the words, are in the form of a proposition; but they are used without any thought as to their real meaning. They are, in effect, but signs of states of feeling, like interjections, and do not help an affirmati or a denial of any quality about anything. The utterance of su interjections as "ah!" "oh!" is certainly no proof that the patie has any power of language in the sense of being able to convey intellectual proposition. The same remark applies to any real wor which the patient utters without being able to *use* them, and evident to the fragmentary jargon some speechless patients utter so copious A patient who had been under Dr. Jackson's care at the Hospi for Epilepsy and Paralysis, and who is still under his observation, c utter such words as "lor," "deah," "me." It would be safer call these fragments "sounds" rather than words, as they are but ra and tatters of talk. They are certainly not what are properly call "parts of speech," and are of no use whatever to this patient in the w of conveying any intellectual meaning. She can utter the wor "yes" and "no," but can hardly be said to *use* them, so that even the are scarcely words to her. Dr. Jackson said that he could never satis himself that she had any power of conveying a proposition, eith by words or even by more simple signs than the conventional soun of spoken words. Latham says, "Without propositions there are questions, commands, or declarations; and without questior commands, or declarations, there would scarcely be such a thi as language. The little there would be would consist merely exclamations like 'oh!' 'ah!' 'pish!'" Max Müller says, "Langua begins where interjections end." And even if language may ha arisen from interjections, according to the theory which Max Müller ca the Pooh-Pooh Theory—and which theory he tries to disprove interjections are not, in the present developed state of language, pa of speech in the sense that nouns or names are. "It is in names says Hegel (quoted by Max Müller), "that we think." If so, it in names only that we can speak. But, although interjections a analogous sounds are not parts of intellectual language, they a important parts of the framework of emotional language, and th help the proposition in a subordinate way.

Here Dr. Jackson referred to Mr. Herbert Spencer's essay, "T Origin and Function of Music," to which essay he was, he sai indebted for anything of value he might have arrived at as regards t distinctness of intellectual and emotional language, and their relatio to one another. The following quotation gives but an incomplete id of Mr. Spencer's views, but it will serve our present purpose: " speech is compounded of two elements, the words and the ton in which they are uttered—the signs of ideas and the signs of feelings. While certain articulations express the thought, certain vocal sounds express the more or less of pain or pleasure which the thought gives. Using the word *cadence* in an unusually entended sense, as comprehending all modifications of voice, we may say that *cadence is the commentary of the emotions on the propositions of the intellect.*"

The jargon the poor woman could utter, although of no use in conveying her ideas, helped her to show her varying states of feeling. Indeed, in this instance, intellectual language was nearly, if not entirely wanting, whilst emotional language was quite perfect: moreover, in this instance, the latter was highly developed. She could easily show that she was pleased or vexed: but the cause of her pleasure or vexation could only be guessed at.

When excited she could vary her tones in the most remarkable way, and would say "ah! ah! me! me!" in the most violent, or at times in the most plaintive, manner. It may be just remarked, in passing, that her emotional expression was in great part natural to her, and was not of that sort one so often sees in cases of softening and of extensive brain disease generally. She had always, her husband said, been excitable and fond of excitement. She would, before her illness, at any time get up in the night to go out to a fire.

Now it will be observed that some speechless patients can sing. Dr. Jackson did not allude to this fact in order to show that voice was unaffected when speech was affected, as, so far as he knew, there was no a priori reason to expect that voice would be lost when speech was lost. The distinctness of voice from articulation is well recognized, and with loss or defect of speech from disease of the hemisphere, aphonia never occurs, so far as Dr. Jackson has observed. The difficulty of articulation from paralysis of the tongue and palate which occurs with aphonia from paralysis of the vocal cords clearly depends on disease in the medulla oblongata, and is quite a different thing. It is only mentioned here in order that it may be expressly excluded. It is needless to say that deaf-mutism is a kind of loss of power to talk which is not considered in these remarks.

There is now attending at the Hospital for Epilepsy and Paralysis a boy suffering from epilepsy, aged 3, who can only say the words "mam, mam," and "dad, dad." It was evident from his general conduct that the boy's mental condition was much below par. Dr. Jackson remarked this to the child's mother, as it was very desirable that the real state of things should be recognized in order that the child might have proper training The poor mother said eagerly in reply, "But he has such a wonderful idea of music." She averred that he could soon learn to hum any tune he heard his father play on his flute. He would, however, never hum a tune when he was told to do so, nor, indeed, would he do as he was bid at any time, partly from inability, but also from wilfulness. Dr. Jackson has also seen another boy who had had loss of speech after attacks of epilepsy or epileptiform convulsions, and who it was said could sing, although he could not talk. As, however, in neither of these instances had he heard the child sing, he would again take the woman's case as an illustration. She could sing, and readily sang a song her husband told her to sing "about Boney party," using the sounds "lor," "deah," "me," instead of words. She varied her voice properly.

Still, having reference to Spencer's views, it seems, Dr. Jackson said, that we may conclude that our muscles may be used in two kinds of language, one intellectual and the other emotional. But the muscles may, in some cases of disease of the hemisphere, be readily put in action for most purposes, when they cannot be used to make signs by words or by pantomime. Although this woman's vocal and articulatory muscles are quite unimpaired, so that she can smile, laugh, chew, eat, drink, swallow, cough, sing, &c., she cannot repeat words said to her, and cannot in any way—putting words out of the question—make signs by her lips, &c Again, although she can gesticulate, and does so frequently, she cannot make signs with her hands, or at the most only with very great difficulty. The following extract from Dr. Jackson's notes gives a good illustration of how well she can use her muscles in varying emotional conditions. It ought to have been mentioned that this patient had had hemiplegia of the right side at the date of the first attack several years ago. From this, however, she soon recovered.

"In order to develop her gabble, her husband said: 'Go and talk to that bird.' She went to the cage, which was hanging from the ceiling in one corner of the room, and, standing up, cried: 'Ah! ah! O deah! deah! deah! Pittymy, pittimy. Lor, lor, lor,' &c. She seemed quite delighted with her task, and varied her voice wonderfully, uttering one set of the gabble in one tone, and the others in other tones. At the same time she gesticulated incessantly, throwing her arms up and down, seeming to accompany her voice with a sort of dance of the arms."

These rhythmical movements are of importance in their relations to cadence. Spencer says: "A smile, which is the commonest expression of gratified feeling, is a contraction of certain facial muscles; and, when the smile broadens into a laugh we see a more violent and more general muscular excitement produced by intenser gratification. Rubbing together of the hands, and that other motion which Dickens somewhere describes as 'washing with impalpable soap in invisible water,' have like implications. (Was it not Hood who said 'washing his hands with invisible soap in imperceptible water?') Children may often be seen to 'jump for joy.' Even in adults of excitable temperament an action approaching to it is sometimes witnessed. And dancing has all the world through been regarded as natural to an elevated state of mind."

The woman would dance when a barrel organ was played in front of her house. Thus, then, she could use her laryngeal muscles not only to utter single sounds like "ah! oh!" but also in the complex process of singing. Again, she could not only use her hands in simple gesticulations, but could use her legs in the more cultivated movements of dancing.

There was no reason to suspect that this patient was hysterical. Dr. Jackson said he had no reasonable doubt that there was disease of the convolutions near the left corpus striatum. Perhaps the corpus striatum was itself somewhat involved, but not very much, as the hemiplegia had been but transitory.

NOTES ON THE PHYSIOLOGY AND PATHOLOGY OF LANGUAGE.

[*Medical Times and Gazette*, June 23, 1866, vol. i, p. 659.]

REMARKS ON THOSE CASES OF DISEASE OF THE NERVOUS SYSTEM, IN WHICH DEFECT OF EXPRESSION IS THE MOST STRIKING SYMPTOM.

THERE are two modes of expression, one emotional and the other intellectual. By one we show what we feel, and by the other we tell what we think. We may show that we are surprised, angry, grieved, or astonished by the former, but by the latter we give to another by signs (generally words) a notion of our thoughts on things. Now, in some cases of disease of the hemisphere (always, in my experience near to and involving the left corpus striatum), intellectual expression is wanting, and emotional expression is well preserved. But we may make the statement broader still, at least in some cases. In some cases of defect of speech the patient seems to have lost much of his power to do anything he is told to do, even with those muscles that are not paralysed. Thus a patient will be unable to put out his tongue when we ask him, although he will use it well in semi-involuntary actions, e.g., eating and swallowing. He will not make the particular grimace he is told to do, even when we make one for him to imitate. There is power in his muscles and in the centres for the co-ordination of muscular groups, but he, the whole man, or the "will," cannot set them a-going. Such a patient may do a thing well at one time and not at another. In a few cases the patients do not do things so simple as moving the hand (i.e., the non-paralysed hand) when they are told. This sort of difficulty generally, however, clears up long before power to talk returns. There is in health a great difference in the power individuals have to do things they are told to do, e.g., to draw in their breath.

I wish here to urge on students who are studying cases in which defects of expression are the most striking symptoms, to work at them from healthy language and through slight defects down to total inability to express anything in any way. Again, we must think of them as defects of mind, as well as of that part or phase of mind which enables us to think aloud in words. And we must never forget that in disease of the hemisphere we may have now and then, besides mental symptoms, amaurosis, epileptiform seizures, bilious vomiting, retracted belly, congestion of the lungs, constipation, and irregular pulse; for the brain is not only the "organ of mind," but it is the nervous system of the nervous system. It need scarcely be said that words, especially such as "mind," "sensory," "motor," &c., fetter our thoughts as well as define them.

Although the difference in the execution of voluntary and involuntary movements is very striking in some cases of loss of speech, the degree of loss of power to utter words must not be taken as a certain index of this difference. The difference probably depends on the difference in quantity of brain damaged near to (as well as in the exact relation of the part damaged to) the highest part of the motor tract, the point of emission of the orders of the "will" to muscles and to centres for muscular groups. Anyone who has tried to use the ophthalmoscope, in many cases where speech is lost or defective, will find how great the difference is in different cases. Thus one of my patients in the London Hospital had power only to say "yes" and "no," and yet the examination of his eyes was an easy matter. He has since rapidly recovered, and soon regained power to make signs for what he wanted. He had lost power to execute, not all movements, but those movements which are most artificial. Dogberry believed that reading and writing (and doubtless talking, although he did not say so) came by nature, but I suppose everybody else believes they are acquired by education. This patient cannot write, but he can copy correctly from a book with his left hand.

A few months ago a patient came under my care who could only say "pooh! pooh!" and an examination of his eyes was almost impracticable. He made efforts, but he never did what I told him, whether it was to look in a particular way or to keep his eyes still. Instead of opening them, he opened his mouth, or screwed up his face, or shut his eye, and could not be got to look in any particular direction, although he seemed to be on the alert to act, and was all the time doing something with his muscles. In this case I have little doubt that more of the brain was affected, and more of the corpus striatum than in the other patient. This patient is almost as bad now as he was six months ago, and still never does anything I ask him to do.

In a third patient who (and who, by the way, is hemiplegic on the left side, and now has much recovered speech) was unable to talk, the ophthalmoscopical examination was at first almost impracticable. Now, it may be said that the patients do not know what we wish them to do. But the last-mentioned patient, when told to "look at my finger," seemed to know what was wanted, for when I was about to give up in despair, he took hold of my little finger as I guessed, deprecatingly, and as much as to say he knew what was wanted, but could not do it. There can, however, be no certainty in this instance. It may be my fancy. But it will be observed that a speechless patient who cannot put out his tongue when told will sometimes actually put his fingers in his mouth as if to help to get it out, and yet not infrequently when we are tired of urging him, he will lick his lips with it. Now, as a rule, the worst of these patients can generally smile, and all such involuntary processes go on well enough. I say generally, for a physician who has seen many cases of defective speech will be obliged to use such words as "generally." I can call to mind but one speechless patient who did not become able to smile; but this patient (now under my care) never does anything but look about him, and eat and drink, and utter the word "yes" when anything is said to him. He will, however, cry "Oh!" and grin when his rigid (right) paralysed arm is extended. He takes his food when it is put near him; but when the nurse (at my request) "forgets" to give him his breakfast, he never makes any signs for it, although he follows her about with his eyes. He will, however, I have little doubt, begin to smile soon. He has only been ill a month. These patients can do things which require scarcely any, if any, education to do, or for which, in other words, the centres exist ready to co-ordinate muscles. It would seem that the centres for the emotional and the semi-voluntary and involuntary actions are in the pons, medulla oblongata, and spinal cord, but where the power is that sets them a-going is not clear.

There are plenty of facts to show that in disease of the corpus striatum those muscles less under the control of the "will" escape, and that through this, the highest part of the motor tract, we are able to direct our *limbs* voluntarily. The views of Mr. Dunn on the relation of defects of speech to disease of the corpus striatum are, I think, of great value in this connexion. Again, there is no more difficulty in supposing that there are convolutions near the corpus striatum for superintending those delicate movements of the hands which are under the immediate control of the mind than that there is one, as Broca suggests, for movements of the tongue in purely mental operations. The phenomena of chorea, I believe, point to this conclusion.

I must here say that I believe less in some of the views propounded by Broca than I did, although I think the scientific world is under vast obligation to him for giving precision to an important inquiry. Yet I cannot but think that my disagreement with M. Broca, as well as many of the differences of opinion on this subject, are to a great extent due to different ways of putting the same thing. (See quotation from M. Broca's pamphlet.) Indeed, I think the evidence I have collected goes to confirm many of his statements as to facts, but I have for some time ceased to go with him in a few of his inferences. (See [1].) I think, then, that the so-called "faculty" of language has no existence, and that disease near the corpus striatum produces defect of expression (by words, writing, signs, &c.), to a great extent, because this is the way out from the hemisphere to organs which the will can set in motion. Hence, as I believe, disease of the convolutions near the corpus striatum is the cause of chorea, which, as regards the limbs, is not so much a disorder of mere motion as disorder of those movements which are voluntary and educated, or at least co-ordinated.

On the education of voluntary movements Bain has some most valuable remarks, and with reference to his views it is not unimportant to draw attention to the fact that chorea is a rare disease either before these movements have been learned or after they have been fully acquired. Unilateral epilepsy seems to replace, and sometimes, I think, to displace, chorea, and both run into actual hemiplegia occasionally.

So much for the superficial distinctions as regards the nature of (and seat of the centres for) voluntary and involuntary motion, and now for the resemblances of the two.

It may seem to be a wilful contradiction, but we find that a patient *does* things involuntarily which he cannot do voluntarily. At all events, these so-called speechless patients give utterance not merely to words, but to words in a relation which gives them at least the appearance of language. I have elsewhere spoken of swearing and of other similar ejaculatory sentences. These seem to border on emotional actions, or, as I have already put it metaphorically, "oaths are phrases which emotion has filched from the intellect to express itself in a more definite way than it could do by mere loudness of tone or manner." We scarcely say anything when we swear, although we utter words in relation. The secret of the perfect utterance of these phrases, which in "speechless" patients are obviously not voluntary, is they are easily elaborated by long habit, and can thus be brought out by a slight stimulus. Such facts seem to me to be of great importance as illustrating Laycock's doctrine on the reflex function of the brain. Indeed, the study of swearing, in spite of the nastiness of this habit, is one of the very greatest interest and importance, in our attempts to trace the graduations of motion, thought, and language.

But I now wish to speak of utterances which are, perhaps, of the same category as the others, but which seem to be rather expressions of the whole mind, as thinking of particular things, and yet which, contradictory as it may seem, the mind cannot get out when it likes. It will be found that patients who usually cannot talk will sometimes utter a real sentence. Laycock (" Mind and Brain," vol. ii, p. 433) has some most important observations on this subject. He says (and this, I think, helps to show the continuity, with differentiation, of these utterances with emotional utterances like interjections) that "a person, usually speechless, under violent emotions may speak. A similar thing is observed with regard to laughter. A person with local disease cannot laugh voluntarily, but he can involuntarily." We had at the Hospital for Epilepsy and Paralysis a patient who could not keep from laughing, even when asking for help in his distress.

The patient, who utters the sound "pooh, pooh!" never said a single word to any of the nurses or students of the hospital for some months, yet his son assures me that his father once, by saying the word "master's," told him where his tools were, and that he once said, with difficulty of articulation, but with intelligibility, " How is Alice getting on?" Although I have always given place to such statements by patients' friends, I confess that I have more than once almost felt that I was giving place to error in recording in my notes statements made to me about the occasional utterances of patients who would never speak to me. I have recorded (see *Royal London Ophthalmic Hospital Reports*, vol. iv, part 4) the case of a "speechless" patient who was said to have one day asked for chops, although I confess that I did not then believe it. I do now. My past unbelief was brought to my mind forcibly the other day. A most intelligent student gave me the particulars of the case of a patient who died with disease of the left hemisphere. Soon afterwards I was talking to this gentleman of such utterance as I have mentioned. He remarked to me that this patient's wife had, with indignation at his (the student's) incredulity, affirmed that her husband, otherwise always speechless, said, " God bless you, my dear." Now, my friend was quite justified in being very incredulous, but I ventured to tell him that it was better to record such statements, although on the authority solely of the relater.

Dr. Martin, of St. Bartholomew's Hospital, has had the great kindness to allow me to mention a case of loss of speech which occurred in his practice. It is the more important, since Dr. Andrew tells me Dr. Martin clearly located the disease, and predicted its nature during the man's life. I have Dr. Martin's permission to make use of the case, but the following quotation from the notes I got from the patient's wife will serve my present purpose: "She told me the following as the only words she had heard him say besides 'No,' since he lost his speech (March 8) to his death (May 15): 'Five nights, six nights, seven nights, and then five nights out of seven.'" These words she assured me he spoke very clearly indeed, and she fancied he wanted to tell her something, but what it was she could only guess.

In nearly all the cases recently observed of loss of speech with hemiplegia the paralysis has been on the right side of the body, indicating disease of the left side of the brain. To explain this, Dr. Moxon suggests that the left side of the brain only is educated, although there is "an organ of language" on each side. I believe this hypothesis is essentially a most important one, but I differ from it in one way, although, of course, I can be but speculating. I think both sides are probably educated, but that the left is the one that begins to act, and that those educated utterances, which are in a sense involuntary, have become so by habit—i.e., frequent education—and may be the result of action of the right side only. How from the right side they are set a-going I do not know any more that I know how a person laughs, not only without effort, but even when he tries to keep quiet.

M. Baillarger has already, I find, considered this question in an admirable manner. He says: "L'analyse des phénomènes conduit à reconnaître, dans certains cas de ce genre, que l'incitation verbale involontaire persiste, mais que l'incitation verbale volontaire est abolie. Quant à la perversion de la faculté du langage caractérisée par la prononciation de mots incohérents, la lésion consiste encore dans la substitution de la parole automatique à l'incitation verbale volontaire." In the following sentence apropos of a particular case, he speaks more generally: "Il est bien évident qu'ici l'incitation motrice volontaire était abolie et que l'incitation motrice spontanée persistait." M. Baillarger refers to cases recorded in Dr. Forbes Winslow's remarkable and valuable work "Obscure Diseases of the Brain."

M. Baillarger has also made some very important observations on incoherence in cases of loss of power to talk voluntarily. It has occurred to me to try to produce an artificial incoherence in "speechless" patients by partial chloroform narcosis. M. Baillarger quotes from Gratiolet a statement to the effect that the frontal convolutions on the left side are in advance of those on the right in their development. Hence, if this be so, the left side of the brain is sooner ready for learning. It is the elder brother.

At first glance there seems to be an utter difference betwixt a smile and such an ejaculation as an oath, and a greater still betwixt a smile and such a sentence as "How is Alice getting on?"[1] There may seem to be an equally great difference betwixt ataxy of articulation and uttering wrong names for things. I have already, in some remarks on a case of chorea, referred to this subject (*Medical Times and Gazette*, January 28, 1865). "It is, in some classes of cases of disease of the nervous system, hard to say where obviously motor symptoms end and where the purely mental ones begin. Thus there is (in cases of hemiplegia on the right side) every gradation betwixt, on the one hand, a total loss of power to express ideas, or a loss of knowledge of the relation of words to things, and, on the other, apparently scarcely more than an ataxy of articulation. And sometimes in the same case we find that the patient makes mistakes in words, and also articulates badly." It is not difficult to show that ataxy of articulation and so-called loss of memory for words are really defects of the same kind, and that the loss of the sign the speechless patient had for a thing is the loss of power to reproduce in his organs (in health from his brain, through series of centres, to the end of his tongue), the *movements* he has learned for that sign, or, at least, the "motor impulse," and that damage near the corpus striatum affects language and thought, not because any so-called faculty resides there (or anywhere, except in the whole brain or whole body), but because more or less of parts which help in making symbols are broken up. The fact that people do not put their tongues in motion when they think may seem to be a great difficulty; but I hope to show that it is not so great a one as is imagined. This will be but a particular expansion of the views which Bain has long taught, and which, indeed, he has applied to speech. "When we recall," he says, "the impression of a word or a sentence, if we do not speak it out, we feel the twitter of the organs just about to come to that point. The articulatory parts—the larynx, the tongue, the lips—are all sensibly excited; a *suppressed articulation* is, in fact, the material of our recollection, the intellectual manifestation, the *idea* of speech." The italics are in the original.

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[1] I would here refer the reader to Mr. Herbert Spencer's "Principles of Psychology," and especially to the chapters "The Growth of Intelligence," "Reflex Action," "Instinct," and "Memory." The facts related in this paper seem to me to be in harmony with certain views Mr. Spencer has put forward in those chapters.

M. Broca makes the following remarks, which amount, in principle, nearly to the same thing, in a pamphlet he was good enough to send me a year or two ago. (Extrait des Bulletins de la Société Anatomique, juillet, 1863.) "Pour expliquer comment un aphémique comprend de langage parlé, sans pouvoir cependant répéter les mots qu'il vient d'entendre, on pourrait dire qu'il a perdu, non la mémoire des mots, *mais la mémoire des moyens de co-ordination que l'on emploie pour articuler les mots.*" The use of the word memory in the sense of its being a distinct faculty is, I think, likely to lead to some confusion. Spencer says, ". . . such a succession of states (*motor impulses*, the results of conflicting impressions) constitutes *remembrance* of the various motor changes which thus become incipient—constitutes a *memory*" . . . "Thus then the nascent nervous excitements that arise during this conflict of tendencies are really so many *ideas* of the motor changes which, if stronger, would cause a *recollection* of such changes, and thus memory necessarily comes into existence whenever automatic action is imperfect." According to this definition, memory is the obtruding of some of the motor impulses on the consciousness, but I suppose M. Broca means by memory of words the connexion the seats of motor impulses (co-ordinating centres, or whatever they may be called) for words have with that other part of the nervous system (whatever and wherever it may be) which set them a-going.

Dr. Fournié has also written admirably on this subject, and indeed on the whole question of language. I follow him in principle, although I differ from him in some details, which, however, I daresay he may not think to be details. I must, however, leave this part of the subject for the present, and take it up from the point of the relation of sensation to motion, and of each of these to centres.

That the convolutions near the corpus striatum have to do with guiding muscles in articulation, there can, I think, be little doubt, and we may make the subject of study wider by inquiring (which I think we may do in the case of chorea) if they are not for corresponding actions—not mere movement—of the muscles of the limbs as well. That the corpus striatum is the centre chiefly for the limbs, as apart from the trunk, pathology, I think, leaves no doubt. In this connexion it is important to mention that Laura Bridgman, when she dreamed, "talked" in finger language, and doubtless she thought by signs she made with her fingers (and their brain connexions). As we generally think in the movements of our articulatory organs, or in the motor impulses, to move them, so she "talked" and thought in the way she was taught.

Dr. William Thomson, the present Archbishop of York, writes ("An Outline of the Necessary Laws of Thought") · "Those among the deaf and dumb who have been taught by the pains of an enlightened humanity to converse and to think must use, instead of the remembered words which we employ, the remembered images of hands in the various combinations of finger-speech (here the italics are mine) *as the symbols of their thoughts.* The deaf and blind, taught the names of objects from raised letters, must think, not by associations of sound, but by touch."

Of course, we do not either speak or think in words or signs only, but in words or signs referring to one another in a particular manner, any more than we move single muscles or muscles co-ordinated to a particular end. Indeed, words in sentences lose their individual meaning—if single words can be strictly said to have any meaning—and the whole sentence becomes a unit, not a word-heap.

Dogberry thought such things as reading and writing come by nature, but we now believe that even the easy flow of talk of the most loquacious is essentially an entirely artificial process, and that even (granting there is truth in the pooh-pooh[1] and bow-wow theories) the origin of signs—i.e., words—had little in it that was like anything instinctive. The Archbishop of York says, "In the fullest sense, language is a divine gift, but the power and not the results of its exercise, the germ and not the tree, was imparted." I am quite aware that written words and finger words are said to be signs of signs, but they belong to the same category as language does, in that writing is an artificial process, and distinct, at least in degree, from such actions as smiling, respiration, and gesticulations.

I think it will be found that the nearer the disease is to the corpus striatum, the more likely is the defect of articulation to be the striking thing, and the farther off, the more likely is it to be one of mistakes of words. This is not contradictory to what I have elsewhere said [1] as to the degree of difficulty in expression depending on the *quantity* of brain damaged "about the highest part of the motor tract, the corpus striatum—the point of emission of the orders of the 'will' to the muscles." I think, too (although I have yet but little evidence on the point), that in cases of embolism of branches of the middle cerebral artery, we shall find the difficulty to be in "finding" the right word (i.e., in making the right signs) rather than in the process of manufacturing the signs, and for this reason that convolutions at a distance are more likely to be damaged, and, again, that the damage is widespread, and often slighter in degree. Finally, I think we have good grounds for investigating if the arterial supply is not so *developed* as to *re*-unite in *actions* parts which have been differently cultivated in *function*. What Professor Bain calls an emotional wave, I think of under the fancy of an arterial wave. I think the occurrence of a particular action is probably due to the local stoppage in this wave. I think so because there is, I believe, evidence to show that subjective movements and sensations in disease are due to local debility of nerve tissue, as Radcliffe has long taught. Moreover, we may, I think, on Radcliffe's hypothesis, at least more easily, explain periodicity and intermissions in such diseases as chorea, epilepsy, ague, &c.

The vascularity of the region of the corpus striatum is, I think, a fact of much significance in the wider study of the relations of nutritive life and functional activity. The subjective sensation of smell of colour, &c. (the so-called aura in some epileptiform attacks), I liken to a sort of involuntary memory of past impressions, and the spasm of the muscles of the hand—the so-called aura or partial fit—which occurs in some patients who are liable to unilateral epileptiform seizures to one of motor impulses. The subjective sensations of disease differ, however, from the rememberings of health and the contending actions of the whole of the muscles of the hand—the extensors striving with flexors, and the stronger getting most of their way—differ from particular purposive movements. In disease it is as if a row of keys were struck down together in mere order of continuity, producing not music, but noise; whilst the revived sensations and motions (or motor impulses) of health are like chords formed of a few notes related in harmony. That there must be some process by which one part of the nervous system arouses in the nervous systems of sense and in the various motor apparatus past impressions, there can, I think, be little doubt, and I would suggest that one link in the mechanism of this process may be the contraction of muscular-coated vessels supplied by ganglionic nerves; or, more generally, that within certain limits life and function are in an inverse ratio. Of one thing I feel pretty

ON THE PHYSIOLOGY OF LANGUAGE.

[*Medical Times and Gazette*, September 5, 1868, vol. ii, p. 275.]

DR. JACKSON treated his subject under the following heads :—

TWO KINDS OF LANGUAGE.

Healthy language is of two inseparable yet distinct forms :—

(1) Intellectual, i.e., the power to convey propositions.

(2) Emotional, i.e., the ability to exhibit states of feeling.

The two are separated by disease. It is intellectual language alone which suffers in most of the cases to be described. Emotional language usually escapes altogether.

Intellectual language suffers *throughout*, not only in its most striking manifestation in (*a*) words, but in (*b*) writing, and (*c*) sign-making.

It is the power of intellectual expression by "movements" of any kind which is impaired, those most special, as of speech, suffering most; those of simple sign-making least, or not at all.

Emotional language is conserved *throughout*, not only in its most striking manifestation by (*a*) variations of voice, but in (*b*) smiles, &c., and in its most simple manifestation by (*c*) gesticulation.

Although thus circumscribed by the term "defects of intellectual language," there are within this limit many varieties of defects produced by disease near the corpus striatum. The author never uses the terms "aphasia," "aphemia," &c.

It is easiest to say what they are *not*.

(1) *They are not defects of voice.*

(2) *They are not defects due to mere paralysis of the tongue and other articulatory muscles.*

(3) *They are not owing to any fault in the outward organs of reception.*

The Special Nature of the Defects of Intellectual Language.

The author arranges the cases he has to mention, for convenience of exposition, in two classes. In the first class the author supposes that the sensori-motor processes for speech are more or less *destroyed;* in the second that they are *unstable* from under-nutrition (see [8]).

Class I.—Severe cases *in which the patient is speechless or nearly so, or in which speech is very much damaged.* In the worst of these cases the patient can only utter some one unvarying word or two words, or some jargon.

Class II.—Cases in which there are plentiful movements, but wrong movements, or *plenty of words, but mistakes in words.*

Under Class II, he points out that taking the phenomena of many cases we find evidences of damage to sensori-motor processes, higher or lower in evolution, according to (*a*) complexity of movements; (*b*) width of interrelation; (*c*) number of associations, from ataxy of the grosser movements of articulation to an "ataxy" of movements embodying ideas.

He then considers very generally, and with regard to all varieties of cases, the defects in complementary modes of intercommunication which accompany defects of speech, and takes the opportunity of considering a question asked, *What is the degree of intelligence these patients have?* He expressly guards himself against any implication that language and thought exist separately. The question is not, How is general mind damaged? but. What aspect of mind is damaged?

He considers the mental condition of patients in Classes I and II as regards:—

(*a*) *Sign-making* is least affected, sometimes seeming to escape altogether. He urges that we should distinguish in degree betwixt power to make simple signs which idiots can make, and the elaborate signs — saying nothing of finger language — which people make who are healthy, except for congenital deafness, and that we should observe if the patients can make signs to signify abstract quality as "blackness."

(*b*) *Writing.*—This suffers more or less in nearly every case of defect of speech from disease of the hemisphere, but varies as much as the defect of speech itself does.

Indeed, writing, and we may add reading, is the same defect in another form. For in each we have to reproduce the motor symbols of the words. Written or printed symbols are "symbols of symbols."

The patient who cannot write can usually *copy* writing correctly.

Patients can often sign their own names without copy when they cannot or will not write anything else.

(*c*) Do the patients know what is said to them? It is usually held that "aphasic persons" do. The author thinks they usually do when they are speechless except for some unvarying jargon, i.e., cases in Class I, but that when—cases in Class II—they have free but disorderly utterance so high as mistakes in words they often do not understand, i.e., quickly understand, words said to them.

(*d*) Can the patients repeat words said to them? They cannot in Class I; in Class II they can, with or without blunders.

The author supposes the reason in (*c*) and (*d*) to be:—

That in Class I, the sensori-motor arrangements for speech are *destroyed* in their *lowest* processes by *limited* disease near to, and involving, the corpus striatum. The sensory aspect of the sensori-motor processes of mind is not reached. It is the "way out" which is broken up.

That in Class II, the sensori-motor processes are impaired, but not destroyed, and that the change is not limited to the region of the corpus striatum, but reaches deeper in the brain.

(*e*) They cannot read, but they can—often at least—understand what is read to them.

(*f*) and (*g*) These points are very cursorily considered.[1]

Educated Movements.

The movements of speech are educated movements, and thus differ widely from those movements which may be said to be nearly perfect at birth, such as those for respiration, smiling, swallowing, &c. All the muscles represented in the corpus striatum unilaterally[2] require a long education, and the most special of these are those engaged in the movements of speech, and next those of the arm. The muscles always acting bilaterally, and chiefly represented bilaterally in the corpora striata, are born with their centres for movements nearly perfect. Thus, then, the term "Intellectual Language" merges in the larger term "Special Movements acquired by the Individual," and the term "Emotional Language" in the term "Inherited Movements" (common to the Race). There is a still more fundamental distinction.

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[1] There is nothing to show what these points were.—[EDITOR.]

[2] It is true that the tongue acts bilaterally, and that it is represented on both sides of the brain. But still it differs from other muscles acting bilaterally, in that each of its sides has a distinct representation in its corresponding (opposite) side of the brain. (See Broadbent's paper, *Med. Chir. Rev.*, April, 1866, p. 479.)

The most General Nature of the Defects.

The author first considers Class I. Here the words previously used give way to terms larger still. In its highest or worst form it is a loss of *voluntary* power with conservation of involuntary capability.

(*a*) The author first illustrates by the case of gross movements—e.g., a speechless patient may be unable to put his tongue out, although it moves well in reflex, &c., processes.

He next shows that there is a corresponding difference even in quasi-mental movements. "Speechless" patients sometimes ejaculate involuntarily.

(*b*) It will be found that some of those patients who cannot talk voluntarily can swear. They utter other ejaculations which are of the same category—meaningless for the expression of ideas about things, although useful as vehicles for the exhibition of feeling. They swear or ejaculate when excited, *and cannot repeat the words of the interjectional utterances when they try.*

(*c*) Next he instances ejaculations more appropriate to the circumstances under which they are uttered, and which are a step higher in speciality.

(*d*) Next, and highest, a man usually utterly speechless may get out an actual proposition.

Here, again, taking into consideration the phenomena of many cases, it will be seen that there is, so to speak, an ascent in "compound degree" from utterances, like the common explosive oath, most *generally* related to *general* external circumstances, to actual propositions *specially* related to *special* external circumstances — i.e., until the difference betwixt voluntary and involuntary utterance is lost.

The above-mentioned series of phenomena show, the author *thinks, that there are in "speechless" patients sensori-motor processes for words somewhere, though usually the "will" cannot get at them.*

This somewhere can scarcely be on the *left* side of the brain, for damage of this side has made the man speechless. These involuntary utterances are, the author supposes, the result of action of the *right* side. In other words, he thinks that the left is the leading side, and the right the automatic.

The Will.

He then tried to show the relation of the so-called "will" to the rest of sensori-motor processes, and this time takes his illustrations from the stock words or phrases which the patients always use. First, he points out that it is probable that the stock phrase was the leading sensori-motor process when the brain was suddenly damaged, and speaks of two cases in illustration. A man becomes speechless after hard work at *making a catalogue*, and can afterwards say only "List complete." (Recorded by Dr. Russell, of Birmingham.) Another man receives a wound in the left hemisphere *in a brawl*, and can only say, "I want protection."

He then speaks of Spencer's views on the "will" and, as he believes in accordance with those views, calls the "will" the *leading* sensori-motor process of the moment, there being no such separation as Will *and* Mind.

The Left Side of the Brain the Leading Side, the Right the Automatic.

The author does not think, as Dr. Moxon does (see *Medico-Chirurgical Review*, April, 1866), that the left side of the brain only is educated, but that both are educated. It is certain that damage to the right side of the brain produces no defect of speech in most cases, and equally certain, the author thinks, that the *disease of the left side only cannot prevent the patient getting out words when a forcible circumstance outside himself is in very special relation with the processes for those words.* For he points out that although there is in cases of involuntary ejaculation no prompting by the "will of the patient," so to speak, the occasional utterances are developed with more or less appropriateness to the external circumstances.

Although the cerebral hemispheres are twins, the left may, if we accept Gratiolet's statement, be said to be the first born. It is born with the lead, and thus a patient who has damage to the left side of his brain cannot *initiate* movements in the undamaged right side, either objectively, as in talking, or subjectively as in reading.

The author has recorded a case of loss of intellectual language in a patient who was *left-handed*, but states that in other cases this explanation will not apply, and he admits that there are cases of defect of intellectual expression with *left* hemiplegia which cannot be explained. Some of the patients, however, have been previously paralysed on the right side, although perhaps without any accompanying defect of speech. Still he has never seen a case of disease of the right hemisphere, only as proved *post mortem*, with defect of speech of any kind, but has recorded three cases in which this side of the brain (including Broca's convolution) was diseased without defect of speech. And all are agreed that when hemiplegia occurs with loss of speech, the hemiplegia is *nearly always* of the right side. He has, however, received reports of two cases, one recorded by Dr. Long Fox, of Bristol, and one by Dr. Pye Smith, in which the Broca's region (on the left) was diseased without loss of speech.

Localization.

The author's views on *Localization* have been already so fully given by himself that it is needless to repeat them. In the last (the fourth) volume of the *London Hospital Reports* the author has reported several autopsies on patients who died aphasic.

NOTES ON THE PHYSIOLOGY AND PATHOLOGY OF THE NERVOUS SYSTEM.

[*Medical Times and Gazette*, November 7, 1868, vol. ii, p. 526.]

Language and Thought—the Duality of Mental Processes.

I suggested in my last article ("Hemispherical Co-ordination," *Medical Times and Gazette*, September 26, p. 358), of which the present is a continuation, that the pair Expression and Perception are really two pairs—(1) Involuntary and (2) Voluntary Expression, and (1) Involuntary and (2) Voluntary Perception.[1] I was led to this supposition by a consideration of the phenomena of some cases of speechlessness the result of damage to one (usually the left) side of the brain, or, as I would put it, of the cases of persons who cannot revive words *voluntarily*. What follows is mostly an amplification of the concluding paragraph of the preceding article, and an attempt (see the part on representative perceptive signs) to make clearer what I have spoken of crudely as a double revival of "images," "concepts," &c.

I ought, in fairness, to state beforehand that most of what follows is speculative. The justification of the speculations lies, I imagine, in their leading to a more orderly method of investigating the revelations which cases of disease of the nervous system are constantly making. It is obvious that much of what follows is accepted doctrine, and the only point I wish myself to urge is the *doubleness* of mental processes.

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[1] It is needful to say how the term "perception" is to be understood in this article. *The psychical, like the physical, processes of the nervous system can only be functions of complex combinations of motor and sensory nerves* I have in the former paper guarded myself against being understood to imply that sensation and movement exist separately, although, for simplicity, I speak of them as separate. Moreover, it is not implied that Sensation and Perception are altogether different. A Perception is a complex and yet an orderly development of impressions chiefly received in the past from *many different* sense surfaces; and when I speak of a sensation arousing a perception involuntarily I simply mean that, for instance, a noise or a touch may at any time make *actual* in the higher parts of the central nervous system (and their peripheral connexions) a *unit*, which is at all times potential, formed out of *many* impressions. It is as if, to use a very crude and inadequate illustration, the sound of one letter were to develop, not an auditory image of itself or several repetitions of itself, but the sound of a whole word made up of different letters, of which word the one letter is perhaps the first letter In perception (as the term is to be used in this paper) several sensations, simple or complex, or both, have yielded, so to

I would especially refer to the writings of Laycock, Spencer, Carpenter, and Lockhart Clarke.

Words serve not only in speech but in thought; in the former they are usually actually uttered; in the latter the processes for them are only nascently excited. But I think it is not sufficiently exact to speak of thought as "internal speech."

It is not enough to say that speech consists of words. It consists of words *referring to another in a particular manner;* and without a proper interrelation of its parts a verbal utterance would be a mere succession of names embodying no proposition. A proposition—e.g., gold is yellow—consists of two names, each of which, by conventional contrivances of position, &c. (called grammatical structure in well-developed languages), modifies the meaning of the other.[1] All the names in a random succession of words may, it is true, one after the other, excite perceptions in us, but not perceptions in any relation to one another deserving the name of thought. The several perceptions so revived do not make a unit. We are *told* nothing by a mere sequence of names, although our organization is stirred up by each of them. Now, a proposition is not—that is to say, in its effect on us—a *mere* sequence.

When we apprehend a proposition, *a relation between two things* is *given* to us—is for the moment, indeed, forced upon us by the conventional tricks which put the two names in the respective relations of

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speak, their own personality into the formation of a higher unit which we then distinguish as a perception.

Similarly in expression, the corresponding opposite of Perception, we find in a certain disorder of articulation (from disease of the higher parts of the nervous system, e.g., of the region of the left corpus striatum) that the defect is not of the muscles of the palate alone, nor of those of the tongue alone, &c., nor of all of these *grouped* together. It is a disorder of a complex unit for the whole process of articulation. But I must defer the more complete consideration of these points until I come to treat of "Spectral Illusions," which are, I conceive, the sensory correlatives of those complex motor disorders which border on, or constitute, incoherent talk.

I adopt the expression "thread of consciousness" for those of our always possible sensori-motor processes, which are for the time *actual* and into which sensations or perceptions are continually bringing modifications.

I may here say something on the use of the words "will," "voluntary," &c. Although we "will," in the sense that we do what we like, provided there are no external hindrances, we do not desire as we like (see Spencer on the "Will," "Principles of Psychology," Lockhart Clarke on "Volition," *Psychological Journal*, Nos. 8, 9 and 10). I must use some such word, and I use the words "will," &c., pretty much as "effort," reflection," &c., are used.

[1] The remarks of Waitz, from whom I have borrowed the idea in the above paragraph, are of very great interest in this connexion. The passages are too long for quotation. (See Waitz's "Anthropology," translated by Collingwood, p. 241 *et seq.*).

subject and predicate. We receive in a *twofold* manner, not the words only, but the order of words also.

Within the speaker's mind the complete process must correspond to this double gift. I remarked in the article above mentioned, "there is [internally] something like a reduction of [apparent] relations of sequence to relations of co-existence (i.e., alternate sequence) in the *two* terms of a proposition." His uttered sentence is the result, I conceive, of a double succession of movements (words). There is, I imagine (when we take into consideration what we may suppose to be the full process which ends in the actual utterance), a corresponding double succession of perceptions, too. I tried to show that in the process which leads to outward speech —not necessarily outward, of course, let us say, to propositions—there is a double revival of words—automatic revival—prior to voluntary revival; and that the *automatic* revival occurs first, and on the *right*, and the voluntary revival afterwards on the *left*, side of the brain. Concepts, images, perceptions, or whatever they may be called—I shall use the word "perception" in this paper—develop words automatically in no order, or rather in their own order, on the right side of the brain. Words are, or may be, objectively considered on the left, put into new relations, made into propositions. I wish to speak of the links *between* these two revivals. The links are, I imagine, the perceptions of which the words are the arbitrary signs. I wish also to speak of the links between the revived perceptions. They are the aforesaid revived movements of words. In fact there is, I conceive, an alternate play between the higher "movements" and "sensations" of mind, as there is in the commoner movements and sensations of the body. In the latter, after a sensation has excited a movement, the presumption is that the sensory nerves from the muscles put in action, or perhaps from the skin over them, register the form and degree of the *movement made or making*, and no doubt in such automatic processes as walking these supervising nerves serve also in the excitation of the movement which is to follow in adaptation to the common purpose of the series of movements—helps to determine *its* form and degree. In walking there is no difference, or no obvious difference, in the alternating movements, but in the leading parts of the nervous system the processes are not altogether automatic. If they were there would scarcely be a consciousness.

It is convenient to speak first of the two revivals of perceptions, and next of revivals of words, although, as already said, I conceive the revivals to be *alternate*—involuntary motor, involuntary perceptive: voluntary motor, voluntary perceptive.

It seems plain that there is, at least often—always, I imagine—involuntary prior to voluntary[1] revival of perceptions. To take an instance. When I hear a certain creaking noise, I "see," in spite of myself, a certain room. I should have so *suffered* any time the last twenty years, and may for the next twenty. Here a simple sensation rouses a perception. The creaking sound is merely a particular noise affecting the auditory nerve; but the induced change in this nerve necessarily revives in the brain to which it travels impressions of other things with which, in co-existence or in sequence, it had been often formerly received. Probably the noise always acts on me in essentially the same way, but in very different degrees. When I am thinking of something important, there is either no obtrusion or the *faintest*, or perhaps I should say only the most *temporary*, obtrusion of the awakened perception on the "thread of my conciousness." But were I not strongly pre-occupied or granting that certain circumstances, say the recent death of a friend whom I used often to meet in that room, were *en rapport*, it would take the lead in my mind. I should "will" to dwell on the perception which has been obtruded on me, and for this there would, I presume, be required a *voluntary* revival of the perception—objective consideration—and this second revival would occur, as I speculate, on the right side of my brain. In short, the sensation first acts upon me as it finds me, unawares. I am suddenly its victim, for it brings the room before me in spite of me. The awakened perception may or may not quickly fade out of my consciousness, or it may be revived by effort when the noise has ceased and is perhaps forgotten. Moreover, it must take a place in, so to speak, any "proposition of perceptions" I like to put it in. Or using popular language, I can reflect on it.

To repeat, there is first involuntary revival of perception, afterwards there is or may be voluntary revival. And of the voluntary revival it must be added that the perception, besides being modified, is or may be put in a new order with others. Indeed, it is necessarily modified by being put in a new order, as words are in a proposition. But what is the intermediation betwixt these two different perceptions? They are really different, each being not merely a repetition of the other. It is movement, i.e., the movement of words.

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[1] Although I must speak for convenience of the two sides as being respectively voluntary and involuntary, I do not think there is any abrupt difference. The remark in the text implies the belief that there are all degrees betwixt automatic action and that kind of imperfect action which is the dawn of "will."

Let us see how the hypothesis applies to what follows hearing the word "horse" in a proposition. Horse (the real thing, that is) cannot be considered objectively in the same sense as the room can. Nevertheless, when the word "horse" is said to me I do properly understand what is meant, and I understand sentences of which the word "horse" forms a part, as "horse is swift." The word cannot, it may be urged, revive the perception of anything automatically, as it is not the name of any one thing—of any particular horse. It serves as a sign for all horses, for horses of different colours, for young horses, for old ones, for fat or for lean ones. Still, it revives something. There have long been permanent modifications of my brain which make it possible for another person at any time to *compel me* to have in my consciousness, at least momentarily, unless I am strongly preoccupied, *some* kind of notion of horse. He excites certain changes in the "grooves" of those permanent modifications of my brain which are always with me, part of me. And although what the speaker can bring about directly is only a certain series of vibrations on my auditory nerves, these vibrations are of necessity, in all Englishmen who have seen horses, followed by a notion of "horse." What is the nature of this ever-present modification of my organization, usually sleeping, but any time to be awakened by the noise "horse," to give me a notion of horse? Is it a sensory, or is it a motor process? In other words, is it a potential, i.e., a unit formed out of certain complex sensations, having a *direct* relation to horse, woven from former seeings of more than one horse—or is it an arbitrary (potential) motor sign in itself altogether meaningless—a counter which is revived to serve in thought *instead* of a perception (a spectre) of horse? I think there must be both a motor and sensory process.

It is plain that I have always in my brain a motor sign for horse, for I can say the word "horse" at any time, and no doubt, when I hear the word "horse," the motor process—the word—is the one first revived in my mind by the aural sensation. Its revival is, fundamentally considered, an instance of a particular movement blindly following, by acquired habit, a particular sensation. This motor sign is, or may be, developed *automatically* on the right side of the brain, for it is possible to rouse it in a man who has lost the side of *voluntary* revival of words by disease.[1] But have I not something more in my mind than the mere *motor* sign when I understand a proposition with the word "horse" in it?

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[1] To take an old illustration—the word "triangle." How, it may be asked, can this motor sign in a proposition develop in us the perception of a triangle, of whose shape nothing special is affirmed, when actual triangles are dissimilar—equilateral, isosceles, and

There must, I conceive, be besides a sensory image of some kind—a perception. Moreover, there is, I speculate, a double revival of perceptions in understanding a proposition with "horse" in it.

The first thing in this double process of perception, I presume, is that the automatic motor sign "horse" acts on the left side of my brain as the creaking sound did, and develops that *perception with which it is arbitrarily associated.* [The creak has a direct relation with the perception "room." The noise "horse" has an indirect relation with this arbitrary sensory image horse, through the word "horse."] So far the process is still involuntary. We must stay here to consider what the nature of the revived involuntary perception is.

In the case of the creak the perception of one *particular room is revived,* not a "general notion" of rooms. The creak is then, so to speak, that one room's proper name. But in the case of horse, there being no particular horse in question, what is the nature of the perception revived?

My notion is that we not only think by help of arbitrary *motor* signs (words), but by help of arbitrary perceptive signs, and that we have permanently in our brain organized forms of the latter, just as we have organized forms for the former—movements of words. I will try to illustrate this, saying, however, beforehand, that the illustrations are scarcely parallel to the case of "horse"—at least, not seemingly so.

When I am to think of a place I have once visited, I "see" faintly some part of it which has struck me most, e.g., of Petersfield, the market-place; or of a certain village, the Green, where there are railings under an elm tree. Such fragments stand to me, I conceive, as counters in ordinary thought for the whole place. If I am made to think of a place I have never seen, but have read of, I have still some visions of maps or of names on maps. These perceptive signs are really

scalene? It is hard to analyse one's thoughts on such a matter, but I feel that I myself have a perception of a particular triangle—viz., an equilateral triangle, which, I suppose, serves, automatically, in thought, for what I may call my general notion of a triangle. Suppose the following proposition is in question: "The three interior angles of every triangle are equal to two right angles." I cannot but think that everyone who heard and understood this statement would have a perception of a drawing of that kind of triangle which had, in his experience, been most frequently associated with the word "triangle," or some one triangular figure which had struck him. Suppose the above statement is repeated in detail. "This is true of the equilateral, the isosceles, and the scalene triangles." There would then be developed with each name perceptions of each kind of triangle—with three equal, with two equal, and with three unequal sides.

sensory sign for it, which is really quite arbitrary. Thus, when I think of Berlin, or of America, all I can put before me for these places are quite as arbitrary signs as words are, although each person has his own perceptive signs. It would seem as if anything of the sort would do, for when I think of New York I "see" a picture I once looked at of Broadway. When I read of scenes in "Arctic Voyages," I think of vast hills of ice and water.

Now, I can in each of the above cases, according to the degree of my direct or indirect knowledge of the place or scene, enlarge the simple sign or alter it, or perhaps translate it into some other preception which the circumstances may require. When I have seen Berlin, I shall have a different perception of it; and even now I can at will think of it as an assemblage of houses with streets, shops, public buildings, &c. There is automatic revival followed by voluntary revival—the latter revival being essentially an adoption of the obtruded perception by my consciousness—i.e., putting it in new relations by which it is necessarily modified.

The above-mentioned perceptive signs for single objects are analogous to proper names, and it does not follow that I have an arbitrary perceptive sign for horse, in a proposition referring to no particular horse. It is next to impossible to analyse one's thoughts on such a point, but my impression is that we have ever present in the nervous system an organized form which, when revived, is a shadowy vision of some horse—perhaps of one particular horse with whose shape, &c., we were struck—and this serves with the word horse for "horse" automatically in rapid thought. It is possible—this is the belief of some, at least—that the word "horse" was at the earliest part of our intellectual life the *proper* name of some one horse, the one, possibly, we first saw. The very same word afterwards becomes the sign of our "general notion" of horse. There is some analogy in the sensory part of the mind; for as regards the room, so often mentioned above, its particular perception very often stands to me for rooms in general, and it is a revived image of this very room that I have mostly "in my mind's eye" when I read of domestic life, and I place the characters of tales in it. It has become to me the analogue of a general name.

I can at any time voluntarily "see" a horse. I can "see" a white one, a black one, a fat one, a lean one. But in rapid thought I see no particular horse, for no one is spoken of. I have only my "lay figure" of a horse. I can translate my single permanent perception of horse into the perception of any particular horse I remember.

ABSTRACT OF THE GOULSTONIAN LECTURES ON CERTAIN POINTS IN THE STUDY AND CLASSIFICATION OF DISEASES OF THE NERVOUS SYSTEM.

Delivered at the Royal College of Physicians.

[*The Lancet*, March 6, 1869, vol. i, p. 344.]

LECTURE II.

CONTINUING the subject of the Medical Physiology of the Cerebral Hemisphere, the lecturer gave a condensed account of some of the innumerable varieties of defects of speech which attend diseaes of, and of the hemisphere near to, the corpus striatum on one side, usually the left. He thought, with regard to the patient's mental condition, it was, above all, most important to bear in mind the lapse of time from the seizure, and mentioned cases in illustration. He related cases of *temporary* loss of speech presumably from embolism; in which cases, as in those of permanent loss of speech, there was an inability to write as well as to speak. He thought that specimens of writing from patients so suffering were much needed. In some cases of temporary loss of speech occurring with unilateral spasm the patient could write. He spoke of children speechless from birth, and alluded to the bearing of these cases on the important hypothesis put forward independently, and almost contemporaneously, by Broca and Moxon. He tried to show that defects of speech should be classified on the same fundamental principle as grossly motor defects—into those implying destruction and into those implying instability of nervous tissue; or, in other words, into those in which there is diminution of power to speak, and those in which the defect is disorder of speech—e.g., mistakes in words. He believes the mental condition is different in the two classes of cases.

With a view to support still further the doctrine of localization he holds, he tried to show from the phenomena of some cases of hemispasm (occurring with disease of one cerebral hemisphere), that there is an order of frequency in the parts in which the spasm commences, and pointed out that there is also a "compound sequence" in the march of the spasm. He insisted on the necessity of recording what is seen to take place in the simplest cases of convulsion, and hoped that from analysing the "compound sequences" in these cases, we may learn something of the internal relations given to muscular regions which externally are widely separated. He pointed out that the phenomena of hemispasm show that muscles acting bilaterally are practically *equally* connected with each of the corpora striata—a conclusion which Dr. Broadbent had some years ago arrived at deductively. The phenomena of the severest cases of hemispasm from disease of one hemisphere, however, in which both sides of the body are convulsed, led him to a modification of Broadbent's hypothesis. Thus, taking one side of the brain, the right, the lecturer thinks the muscles acting unilaterally, both of the left and of the right side of the body, are represented in the right side of the brain, but that the muscles of the left side of the body are especially represented there: Firstly, more in quantity, for they are more affected when the hemisphere discharges; secondly, first in time (instability), for they are affected before those of the right side. He thinks that, having, from the study of the effects of want of action and of "over-action" of the corpus striatum, arrived at a better knowledge of the plan of composition of one nervous organ, we may fairly use this knowledge as a means of further investigation. He thinks that Broadbent's hypothesis, and the facts which so strikingly confirm it, help us to a better understanding of the action of *two* hemispheres in mental processes. From a consideration of the phenomena of such cases as that of a man (the left side of whose brain was damaged) who could not speak, but could understand what was said to him, who could not write, but could copy writing, and who might ejaculate—from the consideration of such a case, the lecturer was led to believe that the fundamental defect in loss of speech from disease in one hemisphere is inability to reproduce words voluntarily, because the leading motor part of one (the left) side of the brain is damaged. Involuntary reproduction (understanding words) remains possible, because the automatic side (right) is intact. Thus, reduced to its simplest form, loss of speech corresponds to hemiplegia, in which form of palsy the voluntary movements are lost, the involuntary preserved. In other words, it seems that the "unit of action" of the nervous system is—as we might expect from the organism being a two-sided one—double the unit of composition. There is, however, a difference betwixt damage to the corpus striatum and damage to the hemisphere just above it, viz., that whilst damage

ON THE ANATOMICAL AND PHYSIOLOGICAL LOCALIZATION OF MOVEMENTS IN THE BRAIN.

[*The Lancet*, January 18, 1873, vol. i, p. 84.]

FOR some years I have studied cases of disease of the brain, not only for directly clinical, but for anatomical and physiological purposes. Cases of paralysis and convulsion may be looked upon as the results of experiments made by disease on particular parts of the nervous system of man. The study of palsies and convulsions from this point of view is the study of the effects of "destroying lesions," and of the effects of "discharging lesions."

And for an exact knowledge of the particular movements most represented in particular centres we must observe and compare the effects of each kind of lesion. It is just what the physiologist does in experimenting on animals to ascertain the exact distribution of a nerve, he destroys it, and also stimulates it. Indeed, this double kind of study is essential in the investigation of cases of nervous disease for physiological purposes. For limited *destroying lesions of some* parts of the cerebral hemisphere produce no obvious symptoms, whilst discharging lesions of those parts produce very striking symptoms. By this double method we shall, I think, not only discover the particular parts of the nervous system where certain groups of movements are most represented (anatomical localization), but what is of equal importance, we shall also learn the order of action (physiological localization) in which those movements are therein represented.

I begin by speaking of destroying lesions, and take the simplest case—hemiplegia of the common form from lesion of the corpus striatum. A blood-clot which has destroyed part of the corpus striatum has made an experiment, which reveals to us that movements of the face, tongue, arm, and leg are represented in that centre. This is the localization of the movements anatomically stated. Physiologically, we say that the patient whose face, tongue, arm and leg are paralysed, has lost the most voluntary movements of one side of his body, and it is equally important to keep in mind that he has not lost the more automatic movements. The study of cases of hemiplegia shows that from disease of the corpus striatum those external parts suffer most which, speaking psychologically, are most under the command of the will, and which, physiologically speaking, have the greater number of different movements at the greater number of different[1] intervals. That parts suffer more as they serve in voluntary, and less as they serve in automatic operation, is, I believe, the law of destroying lesions of the cerebral nervous centres. It may be illustrated in the hemiplegic region itself; that limb which has the more voluntary uses—the arm—suffers more.

I have illustrated by a case of hemiplegia of limited range from a lesion of moderate gravity. But from lesions of different degrees of gravity we have hemiplegia of very different ranges, varying gradually from palsy of the face, tongue, arm and leg of one side, to universal powerlessness. Or, physiologically speaking, there are all degrees, from paralysis limited to the most voluntary parts of one side of the body to paralysis of the most automatic parts of the whole body. The movements of the heart and respiration are less frequent, and the temperature is abased (soon after the seizure, of course, is meant). The patient, to put it in the shortest way, *is reduced to a more or less automatic condition*, according to the gravity of the lesion.

It must be added that degrees of hemiplegia are not simple degrees; that is to say, they are not either degrees of more or less loss of power only, nor degrees of more or less range only, but of both. They are compound degrees. For example, if there be paralysis not only of the *most* voluntary parts of the body, face, tongue, arm and leg, but also of those next most voluntary (or, in equivalent terms, of those next least automatic, viz., loss of certain movements of the eyes and head and side of the chest), we find that the most voluntary parts (face, arm and leg) *are very much paralysed*. In other words, the graver the lesion, not only the more are the most voluntary parts paralysed, but the further spread to automatic parts is the paralysis.

From these facts, supplied by cases of destroying lesions of the centre producing *loss* of movements, we may conclude that the physiological order of representation of movements in the corpus striatum is such that action in health spreads from the automatic to the voluntary; or rather (the unit of action of the nervous system being a double unit, a molecule of two atoms) that there is *first* action spreading from the automatic to the voluntary, and then action spreading in the reverse order.[1] The spreading of healthy movements is best illustrated by degrees of "effort," as in lifting weights. There is first fixation of the more automatic parts of the arm, side of chest (and still further in automaticity according to the preconceived degrees of heaviness of the object), before the most voluntary part, the hand, grasps the weight and then lifts it. The heavier the weight, not only the more strongly are the most voluntary parts used, but the further does the movement spread to the more automatic parts. This compound spreading of healthy movement corresponds to the compound degrees of hemiplegia.

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[1] I shall use (and, after the physiological definition, without any pyschological implication) the words "voluntary" and "automatic." It is not to be implied that there are abrupt demarcations betwixt the two classes of movements; on the contrary, that there are gradations from the most voluntary to the most automatic.

[2] Of course the term "hemiplegia" becomes a misnomer when there is universal powerlessness. I shall have more to say of the universal powerlessness which occurs from disease of but one side of the brain when I consider convulsive seizures.

I will try now to show that the physiological order of gross movements applies to the movements of speech. I say *movements* of speech advisedly, as I think the abrupt distinction made in the expressions "loss of memory for words" and "ataxic affection of speech" is arbitrary and misleading. (On this matter more will be said later.)

The physiological order applies to the classification of the whole of the phenomena of cases of so-called aphasia to the positive—the inability to speak; and to the negative—the ability to understand speech. Taking an ordinary case of entire *loss* of speech, we find that the patient has lost the most voluntary form of expression (speech), and has not lost the most automatic (emotional manifestations). We find that pantomime, which, bordering on gesticulation, stands half-way, suffers little. We find that the exception to the statement that the patient is speechless (for he can usually *utter* some one or two words) is frequently the exception proving the rule. He has lost speech altogether, *except the most automatic of all propositions*—"*yes*" and "*no.*" Even these real words are often only of interjectional value; they can often be used only along the emotional manifestations, can be used, that is to say, automatically only. And, curiously, we find occasionally that the patient who can *reply* "No" correctly may be quite unable to *say* "No" when told. Another occasional exception proves the same thing: He may *utter* oaths or other ejaculations when excited, which he cannot *say*, cannot repeat, when he tries to do so. Occasionally he gets out ejaculations of a less automatic character (less general in the sense of being suitable to fewer occasions). Thus he may say "Thanks," "Good-bye," on fit occasions, but not when he tries. In a narrow corner we see the same thing: he may be unable to put out his tongue when he tries, and yet move it well in all automatic operations. But there is a far wider and far more important illustration to be given. Coining the word "verbalizing" to include all the modes in which words serve, we see that there are two great divisions, or rather extremes of verbalizing, one is the voluntary use of words (speech); the other is the automatic use of words as in receiving speech of others. Now in the ordinary "specimen" of loss of speech the former is lost and the latter is intact. The patient cannot speak at all, but understands all we say, on simple matters at any rate. That he cannot write is simply loss of speech in another form. For the physiological reality of speech it matters nothing whether the proposition be uttered aloud or to ourselves; it is enough that certain nervous powers *be excited, and excited in definite order;* if they be strongly excited, there is external speech; if slightly, there is internal speech. So that internal speech and internal reproduction of words are not synonymous; there is a voluntary internal reproduction of words in new and propositional forms (as occurs when we write); and there is an automatic internal reproduction of words in old and acquired forms or in forms given us, as when we receive and understand words in propositions spoken to us.

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[1] That the unit of action of the nervous system is double the unit of composition is inferable from the fact that the whole nervous system is double; this conclusion runs physiologically parallel with the psychological law that all mental operations consist, fundamentally regarded, in the double process of tracing relations of likeness and unlikeness. The lower parts of the nervous system are plainly double in function, and it would be marvellous if the higher parts were not so too The most automatic of the higher movements of the body "practically" constitute a single series, although we see that they are in duplicate. The two sides of the chest act so nearly together in time, and so nearly equally in range, that there is "practically" but one movement But the very highest movements—those for words—are *apparently* in single order too, but for the very opposite reason. It is because we only consider the *end* of word-processes (speech), and neglect altogether the prior automatic reproduction of words. In the double action, of which the second part is speech, there is first, I suggest, the automatic and unconscious reproduction of words. Later in this paper will be given facts which tend to show: (1) that the unit of action of the nervous system is double the unit of composition; (2) that the higher the nervous processes are the more unlike become the two components of the unit of action; (3) that the unlikeness is first in time, one acting before the other, and second in range, one being in stronger action than the other.

This physiological order will, I think, be of great use in the investigation of mental diseases proper. It seems to me to apply, at any rate, to some comparatively simple mental symptoms which occur in general physicians' practice. After some epileptic or epileptiform seizures, the patient becomes strange or outrageous, and acts queerly or violently. My speculation is, that in these cases he is reduced by the fit to a more automatic mental condition. Thus I have recorded the case of a man (*Lancet*, March 18, 1871) who walked eight miles in a state like that of somnambulism. He was subject to fits, beginning by a subjective sensation of a disagreeable smell, and depending on (as, I suppose, *petit mal* always does) changes in the region of the anterior cerebral artery. Now, just as after a fit of unilateral convulsion a patient is often reduced to a more automatic condition, so far as his *physical* state goes—he is left hemiplegic on one side—so I suppose this patient was reduced to a more automatic condition, so far as his mental state was concerned.[1]

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[1] In cases of slow deterioration of brain, the disposition "alters", I fear that it is that the natural disposition has its way, and that our more animal, our more instinctive habits and desires are no longer subordinated. There is reduction to a more automatic condition; there is dissolution, using this word as the corresponding opposite of evolution. The weaker the mind the more do the more automatic desires have their own way In a few cases of intracranial hæmorrhage, the patient becomes violent and swears; resembles the "drunken man" whose "natural disposition comes out"; the condition expressed by the proverb "*in vino veritas*" is equivalent to a reduction to a more automatic condition in which the natural impulses have freer play.

ON THE NATURE OF THE DUALITY OF THE BRAIN.

[*Medical Press and Circular*, January 14, 1874, vol. i, p. 19.]

I GIVE here a résumé of some of the opinions I have expressed during the last seven or eight years on the nature of the duality of the brain.

That the nervous system is double physically is evident enough. This is a very striking fact, but one so well known that we are in danger of ceasing to think of its significance—of ceasing to wonder at it. A truth becomes a truism. The chief significance of the duality lies, I think, in its bearing on what is most fundamental in mental operations—the double process of tracing relations of likeness and unlikeness.

The nervous system, I repeat, is physically double. I wish to show that it is double in function also, and further, in what way it is double in function. I shall speak of the brain only, taking illustrations, however, from the lower parts of the nervous system.[1]

Not long ago few doubted the brain to be double in function as well as physically bilateral; but now that it is certain, from the researches of Dax, Broca, and others, that damage to but *one* lateral half can make a man entirely speechless, the former view is disputed. Thus, Broca and Moxon suppose that but one half of the brain—the left in the vast majority of people—is educated in words, the function of the other half not being developed in words.

Prior to the researches of Dax and Broca it might have been supposed that the brain was double in function in either of two ways: (1) That action of both halves was required in any mental operation; (2) that either half (indifferently) would serve alone. Neither of these opinions can now be held, with regard to words, at any rate. The two halves are not double in function in the sense that *both* are required for speech, since a patient can speak perfectly well when the *right* half of his brain is damaged, in whatever part the damage is, and how extensive it may be. Nor are they double in the sense that the two halves are such exact duplicates that *either* of them will do for speech, since extensive damage in a certain region of the *left* hemisphere will destroy speech altogether.

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[1] I use the word "brain" to include the cerebral hemisphere and the subjacent motor and sensory tract. I use the word "encephalon" to include all parts of the nervous system within the skull. It is convenient to use the word "half"—meaning a lateral half—when speaking of the nervous system, and "side" when speaking of the body. Thus, disease in the left half of the brain produces paralysis of the right side of the body.

Speaking in more detail, we say that, in the vast majority of cases, extensive damage to the brain in the region of the corpus striatum on the left destroys speech, and that equally extensive damage in the corresponding region on the right does not affect speech at all.[1] The reader will observe that there is no expression of opinion as to the very exact part of the brain injury of which produces loss of speech. Whilst I believe that the hinder part of the left third frontal convolution is the part most often damaged, I do not localize speech in any such small part of the brain. To locate the damage which destroys speech and to locate speech are two different things. The damage is, in my experience, always in the region of the corpus striatum; but in this article it will suffice to speak of the half of the brain affected; it is admitted that there are exceptional cases; loss of speech has occurred from damage to the region mentioned on the right, and the region on the left has been damaged without any affection of speech. *But the matter of most significance is that damage to but one hemisphere will make a man speechless.* This no physician denies, so far as I know.

I shall suppose in what follows that loss of speech results from damage to the *left* half of the brain, although it matters nothing for the argument which half it is. It is enough that there is in every man one half, be it right or left, damage of which will make him speechless.

Contradictory as it may appear at first glance, I think the facts of cases of loss of speech from damage to but one—the left—half of the brain show conclusively that, as regards use of words, the brain *is* double in function. But the verysame cases show also that the two hemispheres are not *mere duplicates* in this function. I hope to show two things—(1) that both halves are alike, in so far that each contains processes for words; (2) that they are unlike, in that the left only is for use of words in speech and the right for "other processes in which words serve." We shall afterwards show that these expressions are only used to mark extremes of degree. Anticipating what has to come, we say that the right hemisphere is the one for the *most* automatic use of words, and the left the one in which automatic use of words merges into voluntary use of words—into speech. Otherwise stated, the rig half of the brain for the automatic use of words, the left the half the automatic and the voluntary use. The expression I have used is that the left is the "leading" half for words (speech). now say what we mean here by speech.

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[1] In this article I illustrate by cases of *loss* of speech, not by cases of *defect* of speech, I do this for the sake of simplicity As will be mentioned later in this paper, there are numerous varieties and degrees of defect of speech from different degrees of damage to different parts in the region of the corpus striatum.

Speaking is not simply the *utterance* of words. The u of any number of words would not constitute speech. Spe "propositionizing." To this meaning the term speech must b kept.[1] That the speechless patient cannot propositionize *aloud* is —he never does. He cannot propositionize internally. He can say "gold is yellow" aloud nor to himself. The proof that he speak internally is that he cannot express himself in writing. write in the sense of copying writing, and can usually copy writing characters. Now, if he can speak internally, why does write what he says to himself? He can say nothing to hims therefore has nothing to write.[2]

For its *character as speech* it matters nothing whether the tion be said to oneself or spoken aloud. Anatomically and logically regarded, we say that the *same* nervous processes are co in internal as in external speech. The difference is that the ex of these nervous processes in speaking to oneself is so slig the nerve currents developed do not spread to the articulat vocal muscles; in speaking aloud the excitation is strong, and do reach those muscles. This fundamental similarity and su difference between internal and external speech must be kept mind.

So, then, the speechless patient has lost speech, not only popular sense that he cannot speak aloud, but in the fullest se cannot propositionize in any fashion. If this be really so, we n say that speech is external thought, for there is no essential di betwixt internal and external speech. We speak not only to te people what we think, but to tell ourselves what we think. Spe

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[1] It is evident enough that there is much behind speech; a proposition is but a series of mental processes and a beginning of another series. But these thin concern us just yet.

[2] In cases of *defect* of speech there is *difficulty* in writing. In some cases of defect there remains considerable power of writing. I know that cases of *loss* of speech recorded by eminent physicians, in which ability to write was not lost. The chron this kind that I have seen have been mostly cases of pretended loss of speech. Be is it conceivable that a person who has lost *speech* should be able to express himself i If a person can express himself in writing, he gives proof that he has *not* lost spe must speak internally before we write—before we express ourselves in writing.

part of thought—a part which we may or may not exteriorize. Again, it is not well to say that thought is internal speech, for the man who is speechless (the man who has no internal speech) can think. How well or ill he can think we shall discuss later. His condition results from a unilateral lesion; his left hemisphere is damaged, but his right is healthy, and in that hemisphere there still, I suppose, lie processes for words.

There are two ways in which words serve in thought; speech is but one way, and this, whether it be internal or external, is, speaking physiologically, a function of the left cerebral hemisphere.

Those who do not limit the definition of speech as we have done, would suppose that if a man had lost *speech* altogether (internal as well as external) there could be nothing further to say about *words* in his case; for it is sometimes assumed that words serve *only* in speech. But the cases of persons who have lost speech show, I consider, that speechlessness does not imply wordlessness[1]; for if I say to a man who cannot speak at all, "Gold is yellow" (or anything not difficult[2] or novel to him), he readily understands it. This shows that he still has processes for words in his brain. His "ideas" of gold and yellow are only to be reached through words. If he had not processes for words *in himself*, how could he possibly understand my words? My words revive his words. If they did not, I might as well speak to him in a language he did not understand, or clap my hands.

To coin the word "verbalizing," to include all ways in which words serve, I would assert that both halves of the brain are alike, in that each serves in *verbalizing*. That the left does is evident, because damage of it makes a man speechless. That the right does may be inferred, because the speechless man understands all I say to him on ordinary matters. And yet since the patient cannot repeat after me, even in writing, the proposition he so readily understands (since he can only receive it and cannot give it out), it follows that the word processes which remain in his right undamaged hemisphere are not of the same kind as those by which speech results. In other terms, word processes are not of the same kind in each half of the brain.

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[1] I ought to mention, however, that it has been said that cases of loss of *speech* give proof that thought is possible without words; it being tacitly assumed that the speechless patient is wordless. The speechless man can think, I suppose, because he has in automatic forms all the words he ever had; he will be lame in his thinking, because, not being able to revive words (to speak to himself), he will not be able to register new and complex experiences of things.

[2] In order that a healthy person may *understand* anything very novel or difficult, speech is evidently required—mostly internal, but it may be external. If a person be told anything complex, we may hear him telling it again to himself. This, I suppose, the speechless patient cannot do; but to superficial observation he understands everything. He understands tales read to him, and remembers their incidents.

Let us consider the two different ways in which, as I suppose, words serve in the two halves of the brain. I illustrate by propositionizing and receiving propositions. To receive a proposition and to form one are plainly two very different things. It is true that in *each* case *our own* nervous centres for words are concerned; but when we *receive* a proposition the process is entirely automatic, and unless we are deaf, or what is for the time equivalent, absorbed, we cannot help receiving it. When anyone says to me "Gold is yellow," I am, so to speak, his victim, and the words he utters rouse similar ones in me; there is no effort on my part; the revival occurs in spite of me if my ears be healthy. Moreover, the speaker makes me a double gift; he not only revives words in my brain, but he revives them in a particular order—he revives a proposition. But if I have to say, "Gold is yellow," I have to revive the words, and I have to put them in propositional order. The speechless man can receive propositions, but he cannot form them —cannot speak.

The left half of the brain is that by which we speak, for damage of it makes us speechless; the right is the half by which we receive propositions.[1]

But this is only an imperfect way of putting it. We, as anatomists and physiologists, have to do only with nervous processes for impressions and movements, and their conditions of energizing; and, as before said, we have to bear in mind that the essential thing is excitation of nervous processes. We have not to dwell with exaggeration on *actual* movements from strong excitations. I say movements advisedly.

We have, as anatomists and physiologists, to study not ideas, but the material substrata of ideas (anatomy) and the modes and conditions of energizing of these substrata (physiology). Where most would say that the speechless patient has lost the memory of words, I would say that he has lost the anatomical substrata of words.[2]

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[1] For fear of misunderstanding, let me now remark, that although betwixt propositionizing and receiving a proposition there is the difference that in the former process there is usually actual utterance and in the latter usually only internal revival of words, this is not the most essential difference. The essential difference is not that betwixt the internal and external use of words, for speech may be internal; we can, and constantly are speaking to ourselves. The difference is in, or corresponds to, the voluntary and automatic use of words.

[2] Psychology is the elder science; mental operations were studied before the brain was known to be the organ of mind. Hence, however much we may wish to study the anatomy and physiology of the higher parts of the nervous system without psychological bias, we are obliged, for lack of others, to use words which have psychological implications. The words "voluntary" and "automatic" are such words, but they are also used physiologically.

The anatomical substratum of a word is a nervous process of a highly special movement of the articulatory series. That we may have an "idea" of the word, it suffices that the nervous process for it energizes; it is not necessary that it energizes so strongly that currents reach the articulatory muscles. How it is that from any degree of energizing of any kind of arrangement of any sort of matter we have "ideas" of any kind is not a point we are here concerned with. Ours is not a psychological inquiry. It is a physiological investigation, and our method must be physiological. We have no direct concern with "ideas," but with more or less complex processes for impressions and movements.

When *movements* of words are spoken of, it is not necessarily meant that *actual* movement of the articulatory muscles occurs. As already mentioned, there is in the left hemisphere during internal speech simply a slight excitation of the *highest* of the nervous processes of the articulatory series, which are strongly excited when words are actually uttered. Similarly, in the automatic revival of movements for words in the right side of the brain (as, for example, in receiving speech of others), there is supposed to be a slight excitation. These may be spoken of as nascent movements, or "ideal" movements. Saying nothing of dreaming that we are speaking, of dreaming of objects and of the internal sight of the blind, there is plenty of positive proof from morbid conditions that central excitations give us ideas of movements when there are no actual movements. The most striking is the production of movements, necessarily ideal, of an absent hand by faradizing the stump. The excitations in this case must be central, awakened by a stimulus of the sensory nerves roused into activity by faradism. The excitation of sensori-motor processes for words, even in the right cerebral hemisphere, may be so strong that these words are occasionally actually uttered. Thus, a speechless patient whose left hemisphere is damaged may occasionally swear when vexed. We now, therefore, classify the movements of verbalizing physiologically into voluntary and automatic, which classification corresponds to propositionizing and to receiving propositions. So now I say the right half of the brain is for the automatic reproduction of *movements* of words, and the left the side for their voluntary reproduction.

But here we must mention that this distinction is not absolute; there are nowhere in the body absolute demarcations betwixt voluntary and automatic movements; and there are in health all gradations from the most automatic use of words to their most voluntary use. Let us show some of the steps: (1) Receiving a [illegible]n. (2) Simple and

ON THE NATURE OF THE DUALITY OF THE BRAIN

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THE double conclusion that the speechless man has lost speech (in the fullest sense that is, not being able to propositionize in any way), and that he has not lost the automatic use of words, harmonizes with the general law I have tried to establish as to the effects of all lesions of the cerebral division of the nervous system. The law, I believe, is that the most voluntary or most special processes suffer first and most. It is only the complementary expression to say that by lesions of the cerebral division of the nervous system, *the patient is reduced to a more automatic condition.* Thus, using the word language in an unusually extended sense, so as to make it include all modes of expression (passive and active), we can say that the patient who has lost speech is reduced to a more automatic condition of language. I will illustrate this point at length. In doing so I must take into consideration the whole of the speechless man's condition—what he can do in language as well as what he cannot do—or we shall not be able to answer the question, "What is the condition of the man's mind out of whom, so to say, speech has been taken?" I submit that this question is not put fairly when it is assumed that the *speechless* man is *wordless.*

Since there are all degrees of gravity of lesion—and many varieties in exact position of lesion—it is plain, a priori, that the "varieties" of defects of speech will be unlimited. And so they are. It is most important to bear this in mind. We shall, however, make an arbitrary limit. We shall continue to take for illustration a chronic case of *loss* of speech; we shall take a case such as may be fairly called "an ordinary specimen of *loss* of speech."

The first great illustration is that there is loss of the most (special) voluntary form of language (speech) without loss of more automatic (emotional manifestations). The patient smiles, laughs, and varies the tone of his voice, and may be able to sing. We find that pantomime which stands half-way suffers little, and gesticulation is not affected at all. As I used to put it, there is loss of intellectual language with conservation of emotional language.

The next illustration shows an exception proving the rule. Most so-called speechless patients can utter the words "yes" and "no." (Each of these words is in effect a proposition.) See the great significance of this common exception; the patient has lost all speech except the two most automatic of all propositions. And even these are often not propositional in function; they are often only of interjectional value; they are uttered with variations of voice to show states of feeling, and are then purely automatic. Nay, we have in the case of one of these propositions decisive evidence of the reality of the distinction we have just made. The word "no" may be uttered automatically by a speechless patient who cannot utter it voluntarily; he can *reply* "no" when he cannot *say* "no." I will recapitulate these significant facts.

There are three degrees of use of the word "no." It is used most voluntarily (as speech) when the patient can *say* it when told. It is used more automatically when the patient can *utter* it in reply correctly; and it is used most automatically when it only comes out like an ordinary interjection with states of feeling.[1]

At this stage let us recapitulate. As it were, looked at superficially, the speechless man is seen to have lost the most voluntary or special part of language (speech), and not to have lost the more automatic language of emotional manifestation. He is in this way reduced to a more automatic condition of language. And then, as it were, by looking more closely, we see (*vide supra*) that of the double process of verbalizing he has lost the more special or voluntary half only; for we found that, although he could not use words voluntarily (could not speak), he yet retained the automatic use of words (could understand what was said to him). Here, again, he is seen to be reduced to a more automatic condition of language. Taking this as being his ordinary condition we have now to consider what occurs in his verbalizing series when he is subject to strong stimulation. We shall find that, starting from that extreme of verbalizing which consists of the most automatic use of words, and which is intact, there occur utterances of diminishing degrees of automaticity nearly up to the simplest voluntary use of words (speech) which he has lost. (We shall show this from several cases.) Observe that we use the word "utterance," for the patient cannot speak his utterances (cannot repeat them when he wills); they are brought out of him by strong stimulations, and these stimulations are, in the case of such deeply automatic utterances as oaths, wide bodily states (emotional states).

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[1] Of course, the man who has the first use has the second and third uses of the word. One of my patients who could not *say* "no" could utter it readily in reply to a question, and would, his wife told me, make his children "behave when they were at the the top of the yard" by shouting "no, no, no," in an angry tone. This patient was the subject of clinical remarks to my class at the London Hospital. I told the students that he could say "no." But, to my chagrin, I could not get him to say it. This puzzled me, for I had heard him utter the word scores of times. Soon it occurred to me to ask the preposterous question, "Are you a hundred years old?" Then he replied, "No." But, very strangely, when I said, "Now say 'No' agai[illegible]not.

Under excitement the speechless patient may *utter* other words and phrases; but these also are exceptions proving the rule; they are interjections, simple, as "oh," or compound, as "God bless my life" Oaths and other more innocent ejaculations have, in spite of their propositional structure, no propositional function. No man intends to *say*, nor is believed by others to *say*, anything when he swears at his own eyes.[1] The Communist orator did not really make a blunder when he began his oration, "Thank God, I am an Atheist," for the expression "Thank God" is used by careless, vulgar people simply as an interjection, there being no thought at all about its primitive meaning. Such ejaculations are verbal utterances more automatic than speech, and less automatic than the use of words in receiving speech of others. They stand betwixt the two extremes. Their automaticity is proved by the fact that the patient cannot repeat them; he may swear, but cannot *say* his oath. But we find still less automatic links than these betwixt the two extremes of verbalizing.

It will be found that a patient who is speechless and who may swear will sometimes also utter words which are not merely automatic signs of his then state of feeling, but which have also a flavour of applicability, e.g., "That's a lie." Still the applicability is in such a case not special. Such an expression is used rather as an offensive missile than as a proposition. Nevertheless, it is a little less automatic than an oath—it is a shade nearer to speech.

I now give an illustration from a class of utterances higher still—an utterance under excitement of a less general character, or, otherwise described, nearer to speech—nearer to the voluntary use of words. My friend Dr. Langdon Down told me this very striking case: A woman was paralysed on the right side, and remained speechless for six months, when she died. She could always say "Yes, but you know," but nothing more; there was one day a single striking exception. This was when a child was in danger of falling, and then she cried out "Take care."

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[1] Hence the line. "He knew not what to *say* and therefore *swore*."

Such an expression, however appropriate, has most affinities to emotional utterances; it is still very general, and is applicable to innumerable circumstances.[1]

But the occasional[2] utterances of speechless patients do not stop even here. I had a patient under my care, who said only "Wo, wo," and afterwards, "Pooh, pooh," always doubling the word. This was one of the very worst cases of affection of speech I have seen. His voluntary power in general seemed to be very much impaired. To use a common expression, "I never could make anything of him."[3] Yet I was assured that this poor fellow one day said "better," and that once after his son had been long urging him to say if he knew him the patient got out this sentence, "How is Alice getting on?" More than this, he had told his son where his tools were by uttering the one word "Master's." Although here is but one word, it clumsily conveys a proposition, the other terms being understood. It is the most special utterance I have ever known from a speechless patient. Indeed, this speechless man did almost speak.

These facts show that the speechless man retains, to some extent, the power of uttering words; we ought rather to say that words come out of him automatically on fit occasions. He cannot *say* what he

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[1] I used to receive very incredulously the statements the friends of patients and others gave me about the occasional utterances of speechless persons. In a case I have recorded, I could not believe that a patient from whom I could never get a word ever said "Take me home." My former unbelief was one day brought back to my mind rather forcibly. A most intelligent student, Mr. Frederick Mackenzie, gave me particulars of the case of a patient who died with disease of the left hemisphere. Soon afterwards I was talking to this gentleman of such utterances as I have mentioned. He remarked to me that this patient's wife had, with indignation at his (the student's) incredulity, affirmed that her husband, otherwise always speechless, said before he died, "God bless you, my dear." He had thought it so improbable that he had not mentioned this ejaculation in the notes he gave me. It was, although of most general applicability, a painfully appropriate utterance for a dying man to make to his wife.

[2] It is proper to remark here that M. Baillarger has written: "L'analyse des phénomènes conduit à reconnaître, dans certains cas de ce genre, que l'incitation verbale involontaire persiste, mais que l'incitation verbale volontaire est abolie. Quant à la perversion de la faculté du langage caractérisée par la prononciation de mots incohérents, la lésion consiste encore dans la substitution de la parole automatique à l'incitation verbale volontaire." In the following sentence, apropos of a particular case, he speaks more generally: "Il est bien évident qu'ici l'incitation motrice volontaire était abolie et que l'incitation motrice spontanée persistait." M. Baillarger has also made some very important observations on incoherence in cases of loss of power to talk voluntarily. I do not know, however, that anyone but myself has advanced any hypothesis as to the duality of word-processes, and the relation of the elements of the dual to halves of the brain.

[3] The following illustration will show this: I one day tried to examine his eyes, and therefore told him to look in certain directions. The examination was almost impracticable. He made efforts, but he never did what I told him, whether it was to look in a particular

utters. It is plain that he has somewhere in him processes for words; but the fact that they are uttered only under excitement, and that they are nearly always well-organized formulæ, and that the patient cannot repeat them, shows that they are not speech. Observe the two things: they are well-organized formulæ, and they are only uttered under strong stimulation, which is not the "stimulus of the will," to use a hackneyed expression. We may fairly conclude that they stand betwixt speech proper and the most automatic use of words, as in receiving speech of others. At the very least the evidence seems to me to point to the conclusion that in the speechless man the most automatic use of words is quite intact. The inference is, that in health there is in the verbalizing process, of which speech is all that is apparent, double action.

There is evidence of a still more general kind. I believe that doubleness in the verbalizing series is but one instance of doubleness in all the nervous processes of the organism. I will try now to show this. In the instances to be given I am not about to compare mental operations with physical movements. I wish to compare the *movements* of words with other classes of movements. Such a comparison could not be entered upon if we used psychological terms, speaking of "memory for words," instead of movements for words. I repeat, that we are not about to compare and contrast mental phenomena with physical phenomena, but physical phenomena underlying certain mental phenomena with other and grosser physical phenomena.

direction or to keep his eyes still. Instead of opening them, he opened his mouth or screwed up his face or shut his eyes, and could not be got to look in any particular direction, although he seemed on the alert to act, and was all the time doing something with his muscles. On but one occasion did he get his tongue out, and then after the nurse had put hers out for his imitation.

It is only in the worst cases that voluntary power is damaged in its most general manifestations. But there are a few cases in which the inability to protrude the tongue continues for some months. I have seen a speechless patient who usually sat up in his room, whose face looked intelligent, who was cheerful and merry, and who seemed to understand all that I said to him, who could not put out his tongue when he tried His daughter, a very intelligent woman, remarked that he could put the tongue out, as she expressed it "by accident," and added, as an illustration of her meaning, that when anyone was leaving him he could say "good-bye," but that he could neither put out his tongue nor say "good-bye" when he tried. He could say "yes," and "no" at any time; and, using the lady's expression, could say, "good bye," "well," "never," *by accident*. She further remarked that the patient would sometimes swear. Although a foreigner, he uttered the short explosive word which is so much in favour with English swearers, but he could not repeat the word when he tried. She asked him to utter the explosive sound when I was there. He laughed and shook his head. The facts of this case are important The inability to put out the tongue is a fact of the same order as the inability to speak. It may mislead the inexperienced to suppose that the speechlessness depends on paralysis of the tongue. The tongue, however, is not paralysed, the easiest proof being that the patient swallows well.

If we look at the most automatic of visible movements—those of respiration—we *see* that they are double, although sides of the chest move nearly equally in range, and they move nearly contemporaneously. It would be marvellous if the highest nervous processes differed *fundamentally* from the lower. It would not, perhaps, seem marvellous if we were studying mental operations only from a psychological point of view, for it will be said that mental operations are under a different regime from physical. We, as physiologists, of course must admit that the movements underlying mental operations differ from such movements as those of respiration. But what I do not admit is that they differ fundamentally. The "form" of co-ordination will be the same throughout the nervous system, notwithstanding special differences. If there be double action in one part there will be in another.

Since the movements of respiration are nearly equal in range, and occur nearly if not quite together in time, there seems to be but one movement. "It is a unified double." Thus, these movements seem to constitute, and in one sense *do* constitute, a linear series, but really we see that they are double.

Similarly the movements of verbalizing *seem* to constitute a linear series, but for a very different reason. If we limited ourselves to the voluntary process of verbalizing—to speech—we should have no doubt that there was only a linear series, for the words of proposition are in linear order. In reality, speech is but the result of an earlier process. Before a proposition is uttered, before voluntary use of words, words must have been automatically revived. The double process in verbalizing seems to be linear, because the parts of the two halves of the brain serving in verbalizing do not, like the parts of the nervous system superintending respiration, act equally in range[1] and together in time. On the right half there is faint automatic reproduction of words *before* the stronger voluntary reproduction on the left.

To repeat, the most automatic of the visible movements of the body seem to constitute a linear series, because both sides act equally in range and together in time; the most special movements of the body, those of verbalizing, for example, *seem* to constitute a linear series, because one half of the brain acts faintly and before the other; but in each case there is double action. The lower processes of the body are a near approach to a "dead double"; the action of one side of the chest is practically a duplicate of the other, and occurs at the same time;[1] but in the higher processes the doubleness is marked, because of the two actions one is slighter than and occurs before the other. In each case the unit of action *is* double.[2]

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[1] Strength of excitation of central nervous processes is the equivalent of increase of range of movements. The proper comparison may seem to be as to differences in the expenditure of nervous force in the two halves, As it appears to me, increase of quantity of nervous discharge tends not only to more powerful movement, but to movement of a wider range; it produces a compound effect. An increase and strength of excitation of the highest processes would be, on its psychological side, not only a more vivid conception of the leading idea, but the development of other ideas automatically associated with it, an increase of range.

But, it may be asked, how do you know that speech is preceded by the *automatic* reproduction of words? A priori, the evidence is strong.

That automatic action must precede voluntary action is, I submit, certain. In gross physical operations we see that it is so. We cannot use the hand before we fix the wrist, nor the arm before the shoulder is fixed, and in heavy strains with this limb, the chest is first fixed and the glottis is closed, after a full inspiration. The more automatic muscles must be in action before the more voluntary, or there would be no *point d'appui*. Taking a psychological illustration, we note that we desire before we will.[3]

In the case of speech we loosely remark that a person speaks as he likes; but how comes it that he does like to utter any particular proposition. If I say, "this blotting-paper is red," the words must have been automatically revived before I uttered them, or why should I have said *that*? But saying they are automatically revived is not saying that they revive themselves. I had the images blotting-paper and red in my mind, and these roused the two words automatically before those words could be put in propositional order, before speech was possible.

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[1] The lower processes are, we see, co-existent. I believe that the most fundamental law of developmental education of the mind is the continuous reduction of successions to co-existences. Things, as voice, automatic use of words, and voluntary use of words, which were developed in successive order, come more and more nearly into simultaneous order; this is one aspect of becoming more and more automatic. The child is born with voice, then acquires automatic use of words (being able to receive speech before it is able to speak), and then speaks (probably first appropriating simple speech formulæ of others). After awhile all these progressively added operations occur practically simultaneously in the utterance of conventional phrases, or, at least, in such utterances as "You don t say so?" used interjectionally. Operations occupying many separate units of time come to occur in fewer or in a single unit of time.

[2] Besides, a proposition is not a string of symbols, each word referring independently to that perception of which it is the symbol. The meaning of a proposition is one, not two meanings in juxtaposition.

[3] "That every one is at liberty to do what he desires to do (supposing there are no external hindrances), all admit; though people of confused ideas commonly suppose this to be the thing denied But that everyone is at liberty to desire or not to desire, which is the real proposition involved in the dogma of free will, is negatived as much by the analysis of consciousness as by the contents of the preceding chapter" (Herbert Spencer, "Principles of Psychology," second edition, vol. i, p. 500).

So far we have spoken only of words, but since words have no meaning unless they refer to things, there is a mental process besides verbalizing.

In popular language words are said to contain or to be symbols of ideas. In physiological language we say that the highest sensori-motor processes of the word series have organic connexions with the sensori-motor processes of other series. In illustration, we shall take one other series, that which refers to objects. We shall, for convenience, speak of gross and simple external objects.

In taking for illustration the two senses, sight and hearing, and the movements corresponding,[1] we are illustrating by the two most important elements of the anatomical substrata mind. The greater part of our mental operations are carried on in auditory and visual ideas. In the investigation of what is often called the "physiology of mind," these two senses, or rather the cerebral processes thereto corresponding, are our chief concerns.[2]

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[1] I say "movements corresponding" because I consider it to be erroneous to suppose that the unit of composition of the highest nervous processes is of a kind fundamentally different from that of the lower; it will be a sensori-*motor* process in the highest as well as the lowest. When we actually see a brick there is necessarily a motor element, and in ideally seeing it—seeing it mentally when it is absent—there will be a nascent excitation of the element of the central process corresponding with ocular movement as well as of that corresponding with retinal impressions. The sensory element corresponds to the "secondary," the motor to the "primary" qualities of the brick. As will be mentioned farther on, I think that the anterior part of the cerebrum is the chiefly motor region, and the posterior the chiefly sensory region of the brain. This is almost equivalent to saying that in the highest ranges of evolution the sensory and motor elements of nervous processes are largely separated geographically; but this does not imply that they do not act together, together in the sense of immediate succession I mean, for in the simplest reflex action the movement *follows* the impression. But in the highest processes the move does not, we may suppose, follow immediately. I believe the separation in the highest processes is significant in what is psychologically abstraction. To take a narrow illustration: after seeing a red square and a blue round we can think of a red round and a blue square. We can transpose primary and secondary qualities; we can transpose ocular movement and retinal impressions.

[2] "Mental actions, ordinarily so-called, are nearly all carried on in terms of those tactual, auditory, and visual feelings which exhibit cohesion and consequent ability to integrate in so conspicuous a manner. Our intellectual operations are, indeed, mostly confined to the auditory feelings (as integrated into words), and the visual feelings (as integrated into impressions and ideas of objects, their relations, and their motions). After closing the eyes and observing how relatively immense is the part of intellectual consciousness that is suddenly shorn away, it will be manifest that the most developed portion of perceptive mind is formed of these visual feelings which cohere so rigidly, which integrate into such large and numerous aggregates and which re-integrate into aggregates immensely exceeding in their degree of composition all aggregates formed by other feelings. And then, on rising to what we, for convenience, distinguish as rational mind, we find the integration taking a still wider reach" (Spencer's "Psychology," vol. i, p. 187).

Just as we have in our brains educated processes for a great number of words, so we have educated processes for a great number of objects. The sensori-motor processes of the former are central processes built out of auditory impressions and consequent articulatory adjustments. (These we may label the audito-articulatory series.) The processes educated in objects are central processes built out of retinal impressions and ocular adjustments. (These we may conveniently call retino-ocular.)[1] Thus we learn the word *ball*, by hearing it, and by the consequent articulatory adjustments. We "learn" the object ball by receiving retinal impressions and by the occurrence of consequent ocular adjustments. There is a motor element in each of these series. Since the two were learned together the two learnings grow up in inseparable association. The sensori-motor process for the word "ball" is in such organic connexion with the sensori-motor process for the object ball that when one is developed by excitation the development of the other must follow, provided the excitation of the former be strong enough. This is what I suppose is the anatomical and physiological equivalent of the popular saying that a word "contains an idea.".

The next question is, "What are the relations betwixt these two series, the audito-articulatory and retino-ocular," the process for words and images of objects?

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[1] For simplicity I arbitrarily exclude the other movements of the body which are concerned with or after ocular movements.

ON THE NATURE OF THE DUALITY OF THE BRAIN.

[Continuation *Medical Press and Circular*, January 28, 1874, vol. i, p. 68.]

WE have seen that the speechless patient does understand what we say to him. But we have only traced the process by which he understands so far as the automatic revival of words on the right side of his brain. There must be something further; for if my saying to him, "gold is yellow," only revives those words (no further process occurring within himself) he can receive no *information* at all; for words by themselves have no meaning; they are symbols. But the words revived, next revive in him the images of the things they symbolize, "gold" and "yellow."[1] I reach these images through the automatic revival of words in the right half of his brain. He assents to this statement, but he dissents when I say "gold is black." But in the latter case the same process must occur. He cannot deny that "gold is black" if the images of "gold" and "black" are *not* revived. He *cannot believe* that gold is black, because, never having seen black gold, and having often seen yellow gold, the word "gold" automatically develops the process for the colour yellow; the stronger association has its way.[2] In short, there is intact in the speechless man all the processes he ever had for the recognition of objects (which is putting ideas of objects, if I may so speak, in propositional order). The speechless man has lost speech only. His audito-articulatory series is damaged in one half of his brain, but his retino-ocular is damaged in neither half. Hence, although he cannot *name* them, he recognizes ojbects, he points out in a picture any object which we name; although he cannot read he recognizes handwriting; although he cannot write he can copy writing, and he may be able to play at cards or dominoes. In none of these things is speech (the voluntary revival of words) concerned, and thus, a priori there is no reason to expect that the speechless man should be unable to perform them. For these things the action of *another* series is required, the series for the recognition of objects—the series for putting objects in relation to one another, making "propositions of objects." This series we have called "retino-ocular." If these processes were not intact in cases of loss of speech the patient could not even find his way in the streets, he could not preconceive his way. For when we go from one place to another we must make, so to speak, a proposition of the images of the two places, or of stages betwixt.

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[1] The following quotation is of interest with regard to the "ideal" reproduction of movements Herbert Spencer says: "To remember the colour red is to have, in a weak degree, that psychical state which the presentation of the colour red produces." "To remember a motion just made with the arm is to have a feeble repetition of those internal states which accompanied the motion—is to have an incipient excitement of those nerves which were strongly excited during the motion" ("Principles of Psychology," vol. i, p. 448).

[2] Although in this paragraph I use the word "image" for convenience (meaning thereby the mental "ideal" representation of an object), we must bear in mind that, as is implied in the preceding paragraphs, movement enters into the anatomical substratum of every "image." The anatomical substratum for the (mental) representation of the object brick, contains a motor element as much as the anatomical substratum of the word "brick" does.

Let us state some of the facts in the last paragraph in another way. The speechless man cannot say "brick"; that he cannot say it aloud is plain, but he cannot say it all, for he cannot write it; nor helped mechanically can he "make the word" when large printed letters are supplied to him. He cannot "speak" the word to himself and so he cannot spell it. If, now, I say to him, "Give me a brick," he will do it instantly. The automatic use of words remains, and by these I can reach his images of objects. Some physicians say of such a case, "He retains the *memory* of the word." It will seem a strange assertion, but I do not think he does. To remember, or rather to recollect, a word is to be temporarily conscious of it; the physiological process being energizing of the sensori-motor process. I believe the word we utter is only a sound to his consciousness, not a special symbol, not a sound arbitrarily referring to some thing; but it is a sound which has (the consequent auditory change, I should say) organic connexion with the *automatic* movement for the word "brick," and it is through this *automatic* and entirely unconscious process that the nervous process for the image of the object brick is reached. I do not believe that the man who cannot say (nor write) the word brick can be said to have a "memory" of it (be conscious of the *word itself*). He has no consciousness of *it*, but of the thing it is the symbol of—a very different thing. There is nothing strange in supposing that there is an unconscious use of words. The hypothesis accords with the law that the more operations are automatic the less are we conscious of them; of the most automatic of the bodily operations we are not conscious at all.

We have concluded that the verbalizing (or audito-articulatory series) is double, and now we have to infer, we have, indeed, already implied, that the retino-ocular series is double also. But just as we did not consider the audito-articulatory series to be in mere duplicate so we do not consider the retino-ocular series to be in mere duplicate. It must be admitted, however, that the direct evidence in favour of the doubleness of the latter series, or rather of the nature of its doubleness, is very vague and fragmentary. But there seems to me to be a strong a priori warrant for the assumption that there is in it a doubleness analogous to that in the audito-articulatory series. That there is involuntary revival of images seems to me to be clear, since I can compel any one to have before him an image, for example, of a horse, by uttering the word in his hearing. I do this not only without his aid, but in spite of him. Again, it seems clear that no one can think of an object before it is presented to him[1] before it is revived; it must be automatically revived before it is voluntarily revived ("subjective" action prior to "objective" action). To say that a train of thought leads up to it "spontaneously" is really saying this.

I submit that one half of the brain is for the automatic revival of images and one for their voluntary revival. Before giving the reasons for this hypothesis I would remark, that limiting mental operations to the audito-articulatory and retino-ocular series, the speechless man has thought so far as reviving images go. He has words which can be automatically revived so as to place images of objects in order. Although he has but one side for verbalizing—the automatic side—he has according to my hypothesis, *two* sides for the revival of images, and thus he can still think, can still have certain relations of likeness and unlikeness. No doubt he is lame in his thinking. He will be unable to learn novel and complex things, for he will be unable to keep before himself the results of complex arrangements of images. He cannot speak to himself, to tell himself what he has managed to think of, things presented or represented in very novel and complex relations. He can bring two images into co-existence, existence in one unit of time, but cannot, without speech, organize the connexion, if it be one of difficulty.

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[1] It seems to me that this view clears the difficulty stated under the heading, "Attention," pp. 131 *et seq* of Mansel's "Metaphysics" "It appears certain," Mansel says, "on the one hand, that, in order to arouse the attention of any sensible phenomenon, that phenomenon must first be presented to consciousness; while, on the other hand, it has been argued, with some plausibility, that unless the attention be previously aroused, consciousness has no intimation of the existence of the phenomenon at all." Again, "Is the phenomenon of which we become fully conscious by attention the *same phenomenon* that it was before we attended to it? Or has attention itself added an element which brings it within the sphere of consciousness?"

The reader will have remarked that we use the expression "to recognize" (not "to see"). To recognize a thing is really an act of classification (Spencer). It is really putting the image of it in a "proposition" with another image—the image of an object presented with one re-presented. The speechless man can recognize.

Before, however, we can consider which half of the brain is concerned in the voluntary and which in the automatic revival of images, we must consider in which region of the hemisphere the revival of images in any way occurs. I believe it is chiefly[1] in the hinder part—in the posterior lobe—in other words, in the region of the thalamus opticus. That it is not chiefly in the anterior lobe (in the region of the corpus striatum) of either side is certain, for damage here scarcely ever produces any other mental defect than that of (by damage of the left half) affection of speech. The revival of images, I believe, chiefly occurs in the posterior regions of the brain. Evidence that the function of the posterior lobes differs in some way from that of the anterior, is that Lockhart Clarke has found the intimate structure of the convolutions to differ in the two regions. Difference of structure of necessity implies difference in function. It would seem that the posterior lobes are more important in intellectual operations. Dr. Charlton Bastian concludes an important article ("The Human Brain," *Macmillan's Magazine*, November, 1865, p. 71), by saying: "Still, broad groups of functions may be more intimately connected with particular lobes, and, if such be the case, then we believe the evidence in our possession *points to the posterior rather than to the anterior lobes of the cerebrum as those concerned more especially with the highest intellectual operations.*" (The italics are mine.) The posterior lobes are rarely damaged by any sort of pathological change, and thus it is difficult to settle the question by cases of disease. But the following quotation from Rosenthal strongly supports Bastian's opinion: "In the case of new growths in the posterior lobes the *psychical* disturbances are *incomparably more*

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[1] I say "chiefly" because I do not believe in abrupt geographical localizations. Thus, very sudden and very extensive damage to *any part* of the left cerebral hemisphere would produce *some* amount of defect of speech, and I believe that similar damage to any part of the right hemisphere might produce *some* defect of recognition.

I may here say also that, so far as I have yet seen, the difference in the two sides in the retino-ocular series is not so sharply marked as in the case of the audito-articulatory. Not very long ago I diagnosed that a patient had tumour in his right posterior lobe, on account of a mental affection which was not loss of speech; the tumour was in the left posterior lobe.

Nevertheless, I am convinced that disease of the *right* cerebral hemisphere is more likely to cause mental defect (other than affection of speech) than is disease of the left; and, again, that mental defect is more likely to result the farther back in the hemisphere the damage is.

frequent than in that of tumours of the anterior or middle lobes." (The italics are mine.)

So far we have concluded that the revival of images does not occur in the anterior lobes, and we have brought evidence to show that the posterior lobes are more important than the anterior in intellectual operations. The latter is almost equivalent to saying that the posterior lobes are the parts in which revival of images occurs; for the greater part of our intellectual operations is carried on in images, in eye-derived or, as we call them, retino-ocular processes. On the relative importance of visual ideas I have already quoted Spencer.

I think that the left is the side for the automatic revival of images, and the right the side for their voluntary revival, for recognition. I have two reasons for thinking so. The first is one supported by anatomical facts. The following extract from an article I published [17] contains these facts: "It would seem by certain observations of Gratiolet—which are embodied in the following extract from M. Baillarger's address before the Academy of Medicine—that there is "crossed development of the brain," if we may take the corpus striatum and thalamus as fixing the (chiefly) motor and sensory regions. The first part of the quotation refers to M. Trousseau's views on the possible explanation of the rightsidedness of the paralysis of speechless patients.—

"Deux faits anatomiques ont ici une assez grande importance."

"De ces deux faits, le premier relatif à la circulation a déjà été rappelé par M. Trousseau. C'est que l'artère carotide gauche naît directement de la crosse de l'aorta tandis qu'à droite cette même artère naît du tronc brachio-céphalique.

"Le second fait a été signalé par Gratiolet, ce professeur si eminent, dont la science déplore la perte récente.

"Il m'a semblé, dit-il, par suite d'une série d'observations *consciencieusement étudiées*, que les deux hemisphères ne se développent pas d'une manière absolument symétrique. Ainsi le developpement des plis frontaux paroît se faire *plus vite à gauche qu'à droite*, tandis que l'inverse a lieu pour les plis des lobes occipitaux et sphénoidaux." (Leuret et Gratiolet, "Anatomie du Système Nerveux," p. 241.)

It would seem by this, at all events, that the "important side" of the left hemisphere is the anterior lobe, and the important side of the right the posterior lobe. However, I have to say that Carl Vogt denies the truth of the observation that the left frontal convolutions are developed in advance of those of the right. Whether Gratiolet's statement that the sphenoidal and occipital convolutions of the *right* side are developed in advance of those of the left is denied or not I do not know, nevertheless, his name will have some weight to keep the question an open one. M. Barkow, and after him M. Broca, find "que les circonvoultions sont notablement plus nombreuses dans le lobe frontal gauche que dans le droit, et que *tout à contraire le lobe occipital droit* est plus riche en circonvolutions que le gauche. (The italics are mine.) Carrier ("Etude sur la Localisation dans le Cerveau de la Faculté du Langage articulé"), from whose admirable paper I take this observation, goes on to say, that Broca finds that the *left* frontal lobe is sensibly heavier than the right, and that since the two hemispheres are nearly of the same weight, there must be compensation by difference in the two occipital lobes."

These anatomical facts, I submit, support the view I put forward, that the posterior lobe—or let us speak more generally—the hinder part of the brain on the right side, is the chief seat of the revival of images in the *recognition* of objects, places, persons, &c. The other evidence is supplied by cases of disease, and I may at once say that it is slight and doubtful.

But before we proceed to this evidence we must consider how loss of power to revive images voluntarily—that is, recognize—would appear as a symptom; in other words, what would be the obvious result of damage to the retino-ocular series; in other words still, what in the retino-ocular series would correspond to loss of speech in the audito-articulatory series. Such a condition would be one of imbecility. The condition of a patient who could recognize nothing, would pass as a general, not as it really would be, a special mental symptom. In such a case speech would be affected in a sense, still, in the strict sense, speech would not be affected, i.e., directly affected. The patient would have no motive to speak, he would have nothing to speak about.

In a *minor* degree, such a state would be one which the patient or his friends would call "loss of memory." The patient would have *difficulty* in *recognizing* things; he would have difficulty in relating what had occurred, not from lack of words, but from a prior inability to revive images of persons, objects, and places, of which the words are the symbols. He would not be able easily to objectivize. He could not put before himself ideal images of places one after another; could not re-see where he had been, and could not therefore tell of it in words. This again might be looked on as a *general* mental defect, not as a defect really as special as defect of speech.

It may be well to give an illustration of the kind of defect: A patient who was in the London Hospital supposed that she was at the place in Holborn where she had worked for some years, a place the image of which was in her mind more automatic. It was a case of mis-recognition, analogous to a mistake in uttering words more automatic than those intended.

We come now to evidence from cases of disease. I have noted that in cases of left hemiplegia, especially when the leg suffers more or recovers later than the arm, there is what *seems* at first glance a general loss of mental power, but which, as above said, is probably as special a loss as affection of speech is. In cases of hemiplegia, where the arm suffers much more than the leg, I believe the lesion to be placed in the region of the thalamus, that is, in the region of the posterior lobe.

Trousseau, speaking of those cases of hemiplegia in which the arm recovers more rapidly than the leg does, remarks that the patient is worse off than when the reverse obtains. It is not quite clear that Trousseau refers to failure of intellect. I think he does, for he relates in illustration the case of a man who died in a state of perfect imbecility, and he predicted that the woman whose case was the text of his lecture would die within the year a thorough imbecile. It is important to observe that the man had excruciating pain, and this symptom also Trousseau believed would follow in the woman's case. The late Dr. Bazire, in a footnote to Trousseau's lecture, speaks of the case of a patient who was seen in private practice by Dr. Ramskill for hemiplegia like that Trousseau spoke of. In less than a year this patient died, "completely demented."

But Trousseau says nothing as to the importance of the side affected in these cases. He mentions the side paralysed, it was the right; and so it was in Dr. Ramskill's patient, and as right-sided paralysis is evidence of disease of the *left* hemisphere, I have to point out prominently that these cases are in antagonism to the view I take, namely, that the *right* posterior lobe is the more important one. There were no autopsies, however, and therefore these cases do not tell conclusively against it. In each of the three cases, the imbecility came on some time after the paralysis, and thus it may be said that there was a double lesion, one on each side of the brain. I will not, however, avail myself of this explanation, because, as the imbecility occurred, so far as I know, without any further attack, it is simply begging the question. The defect I speak of is mainly a sensory defect;

and thus it does not follow that (as loss of speech depends on disease of the brain near the corpus striatum), loss of power to recognize depends on changes near to the thalamus; they may be deep in the posterior lobe.

As will be seen, my facts are very few, but I do think that patients who have that variety of left hemiplegia in which the leg suffers more than the arm, have greater mental defects of the kind I speak of than occur in other kinds of hemiplegia on the right or left.

It is important to note the effects of plugging of the posterior cerebral artery, which vessel supplies the posterior lobe, and also (what is significant) sends a branch to the corpora quadrigemina. I have seen but one case of this kind, and as in that case *both* posterior cerebral arteries were blocked, and also the right middle cerebral, the case is scarcely worth mentioning as evidence.

REMARKS ON NON-PROTRUSION OF THE TONGUE IN SOME CASES OF APHASIA.

[*The Lancet*, May 18, 1878, vol. i, p. 716.]

RESPECTING this interesting and curious clinical phenomenon, Dr. Hughlings Jackson remarked :—

It will have been noticed by every medical man that some patients who have loss or defect of speech do not put out the tongue when they are asked. For the present we shall consider cases of *loss* of speech. The patients know what is wanted; they do the preliminary thing—opening the mouth—but very often the tongue lies flat and motionless in the floor of the oral cavity. The patient may put his fingers in his mouth to help the organ out. That the tongue is not paralysed (we mean in the ordinary medical sense of the term paralysis) may be proved in several ways. As a rule, the patient, who can *say* nothing, can *utter* something; he has a stock word or phrase which comes out at any time. This is best called a "recurring utterance." The word or phrase being clearly articulated, is decisive evidence that the tongue is not paralysed. Then there are in some cases "occasional utterances." The patient may swear, or get out more innocent ejaculations as "Oh dear!" These also are clearly articulated. Yet very early in a case of loss of speech there may be no sort of utterance. Then we can demonstrate that the tongue is not paralysed by getting the patient to eat and drink. He will eat and swallow quite well, which he could not do were his tongue paralysed. Again, after failing to put out his tongue when he tries, the patient may stick it out well to lick his lips. In one case an aphasic woman was vainly urged to put out her tongue. Mr. Lewis Mackenzie, the resident medical officer, got it out by a trick; he had found that after drinking she would stick her tongue out to lick her lips; she did so on this occasion.

This is a matter of great importance. In some cases of acute cerebral disease the non-protrusion of the tongue would be of great value in helping to the diagnosis of aphasia, as one symptom of that acute disease. In any case it would be a great blunder to suppose there to be paralysis of the tongue from disease of the lingual nerves or their nuclei in the medulla oblongata, because the patient did not put out his tongue when asked, and when he tried. It is a thing of great scientific interest; considered along with some other symptoms of aphasia, it gives us a clue to the physiology of the whole of aphasia. Let us mention some other symptoms of fundamentally like significance.

Those who have examined the eyes of many aphasics will remember cases in which the use of the ophthalmoscope was very difficult; some aphasics do not, and apparently cannot, direct their eyes as they are told. Of course, in some cases of aphasia, there may be for a few hours or days, or in rare or severe cases for several weeks, lateral deviation of the eyes, from the side paralysed, as part of the hemiplegia; but we just now refer to chronic cases in which there is no ordinary affection of the ocular muscles; the difficulty is not in looking to one side in particular. Indeed, we see the same kind of thing in some people whose nervous systems are sound. Some people are unable to draw in their breath deeply when told to do so during stethoscopic examination; we have to tell them to cough. It is next to impossible to get some patients to frown (as in suspected one-sided facial paralysis), even if we make a frown for them to imitate. Returning to aphasia. The recurring utterance is often "Yes" or "No," or the patient may be able to utter both these words. Some of these patients can utter "No" emotionally, and some can do this, and can also reply with it (use it propositionally). But of those who can reply with it, there are some who cannot say it when told. After failing to get them to say it when told, we can easily get the word out as a reply to a question requiring dissent. Similarly of occasional utterances of less automaticity; a patient may swear well on proper (scientifically proper) occasions, and yet be unable to repeat the ejaculation, or any word of it. The same applies to occasional utterances of still less automaticity, as is illustrated by the following excerpt from an unpublished paper by Dr. Hughlings Jackson, written ten years ago: "I have seen a patient who usually sat up in his room, whose face looked intelligent, who was cheerful and merry, and who seemed to understand all that I said to him, but who could not put out his tongue when he tried. His daughter remarked that he could put the tongue out, as she expressed it, 'by accident,' and added, as an illustration of her meaning, that when anyone was leaving him, he could say 'good-bye,' but that he could neither put out his tongue nor say 'good-bye' when he tried. He could say 'yes' and 'no' at any time; and, using the lady's expression, could say 'good-bye' 'well,' 'never' *by accident.* She further remarked

ON AFFECTIONS OF SPEECH FROM DISEASE OF THE BRAIN.

[BRAIN, 1879, vol. i, p. 304.]

IT is very difficult for many reasons to write on affections of speech. So much, since the memorable researches of Dax and Broca, has been done in the investigation of these cases of disease of the brain, that there is an *embarras de richesse* in material. To refer only to what has been done in this country, we have the names of Gairdner, Moxon, Broadbent, William Ogle, Bastian, John W. Ogle, Thomas Watson, Alexander Robertson, Ireland, Wilks, Bristowe, Ferrier, Bateman, and others.[1] To Wilks, Gairdner, Moxon, Broadbent, and Ferrier, I feel under great obligations. Besides recognizing the value of Broadbent's work on this subject, I have to acknowledge a particular indebtedness to him. Broadbent's hypothesis, a verified hypothesis, is, I think, essential to the methodical investigation of affections of speech. Let me give at once an illustration of its value. It disposes of the difficulty there otherwise would be in holding (1) that loss of speech is, on the physical side, loss of nervous arrangements for highly special and complex articulatory *movements*, and (2) that in cases of loss of speech the articulatory *muscles* are not paralysed, or but slightly paralysed. I shall assume that the reader is well acquainted with Broadbent's researches on the representation of certain movements of the two sides of the body in each side of the brain; the reader must not assume that Broadbent endorses the applications I make of his hypothesis. The recent encyclopædic article on Affections of Speech, by Kussmaul, in Ziemssen's "Practice of Medicine," is very complete and highly original. It is worthy of most careful study.

The subject has so many sides—psychological, anatomical, physiological, and pathological—that it is very difficult to fix on an order of exposition. It will not do to consider affections of speech on but one of these sides. To show how they mutually bear, we must see each distinctly. For example, we must not confound the physiology of a case with its pathology, by using for either the vague term "disease." Again,

we must not ignore anatomy when speaking of the physical b
words, being content with morphology, as in saying that words "
in this or that part of the brain. Supposing we could be certain t
or that grouping of cells and nerve-fibres was concerned in speec
its being always destroyed when speech is lost, we should still
find out the anatomy of the centre. Even supposing we we
that the psychical states called words, and the nervous states
"centre for words," were the same things, we should still ha
anatomy of that centre to consider. The morphology of a cent
with its shape, with its "geographical" position, with the siz
shapes of its constituent elements. A knowledge of the anatom
centre is a knowledge of the parts of the body represented in
of the ways in which these parts are therein represented. W
much has been learned as to the morphology of the cerebrum—c
topography—it is chiefly to the recent researches of Hitzig and
that we are indebted for our knowledge of the anatomy of many
convolutions, that is, a knowledge of the parts of the body thes
volutions represent. It is supposed that the anatomy of the
the brain concerned with words is that they are cerebral
arrangements representing the articulatory muscles in very spec
complex movements. Similarly, a knowledge of the anatomy
centres concerned during visual ideation is a knowledge of those
of the brain where certain parts of the organism (retina and
muscles) are represented in particular and complex combinatio
merely materialistic or morphological explanation of speech or
supposing one could be given, is not an anatomical expla
Morphologically, the substratum of a word or of a syllable is
up of nerve-cells and fibres; anatomically speaking, we say it i
up of nerve-cells and fibres representing some particular artic
movement.

Unless we most carefully distinguish betwixt psychology a
anatomy and physiology of the nervous system in this inqu
shall not see the fundamental similarity there is betwixt the
often described in psychological phraseology as "loss of mem
words," and the defect called ataxy of articulation. A method
is founded on classifications which are partly anatomical and
logical, and partly psychological, confuses the real issues. These
classifications lead to the use of such expressions as that an
a word produces an articulatory *movement*; whereas a psychica
an "idea of a word" (or simply "a word") cannot produce an

latory movement, a physical state. On any view whatever as to the relation of mental states and nervous states such expressions are not warrantable in a *medical* inquiry. We could only say that discharge of the cells and fibres of the anatomical substratum of a word produces the articulatory movement. In all our studies of diseases of the nervous system we must be on our guard against the fallacy that what are physical states in lower centres fine away *into* psychical states in higher centres; that, for example, vibrations of sensory nerves *become* sensations, or that somehow or another an idea produces a movement.

Keeping them distinct, we must consider now one and now another of the several sides of our subject; sometimes, for example, we consider the psychical side—speech—and at other times the anatomical basis of speech. We cannot go right on with the psychology, nor with the anatomy, nor with the pathology of our subject. We must consider now one and now the other, endeavouring to trace a correspondence betwixt them

I do not believe it to be possible for anyone to write methodically on these cases of disease of the nervous system without considering them in relation to other kinds of nervous disease; nor to be desirable in a medical writer if it were possible. Broadbent's hypothesis is exemplified in cases of epilepsy and hemiplegia, as well as in cases of affections of speech, and can only be vividly realized when these several diseases have been carefully studied. Speech and perception ("words" and "images") co-operate so intimately in mentation (to use Metcalfe-Johnson's term) that the latter process must be considered. We must speak briefly of imperception, loss of images, as well as of loss of speech —loss of symbols. The same general principle is, I think, displayed in each. Both in delirium (partial imperception) and in affections of speech the patient is reduced to a more automatic condition; respectively reduced to the more organized relations of images and words. Again, we have temporary loss or defect of speech after certain epileptiform seizures; temporary affections of speech after these seizures are of great value in elucidating some difficult parts of our subject, and cannot be understood without a good knowledge of various other kinds of epileptic and epileptiform paroxysms, and post-paroxysmal states. After a convulsion beginning in the (right) side of the face or tongue, or in both these parts, there often remains temporary speechlessness, although the articulatory muscles move well. Surely we ought to consider cases of discharge of the centres for words as well as cases in which these centres are destroyed, just as we consider not only hemiplegia but hemispasm. Before trying to analyse that very difficult symptom called ataxy of articulation, we should try to understand the more easily studied disorder of co-ordination, locomotor ataxy, and before that, the least difficult disorder of co-ordination of movements resulting from ocular paralysis. Unless we do, we shall not successfully combat the notion that there are centres for co-ordination of words which are something over and above centres for special and complex movements of the articulatory muscles, and that a patient can, from lesion of such a centre, have a loss of co-ordination, without veritable loss of some of the movements represented in it.

It might seem that we could consider cases of aphasia, as a set of symptoms at least, without regard to the pathology of different cases of nervous disease. We really could not. It so happens that different morbid processes have what, for brevity, we may metaphorically call different seats of election; thus, that defect of speech with which there are frequent mistakes in words is nearly always produced by local cerebral softening; that defect which is called ataxy of articulation, is, I think, most often produced by hæmorrhage. Hence we must consider hemiplegia in relation to affections of speech; for it so happens that the first kind of defect mostly occurs, as Hammond has pointed out, without hemiplegia, or without persistent hemiplegia, a state of things producible by embolism and thrombosis, and the latter mostly with hemiplegia and persistent hemiplegia, a state of things usually produced by hæmorrhage. From ignoring such considerations, the two kinds of defects are by some considered to be absolutely different, whereas on the anatomico-physiological side they are but very different degrees of one kind of defect.

There are certain general principles which apply not only to affections of speech, but also to the commonest variety of paralysis, to the simplest of convulsive seizures, and to cases of insanity.

The facts that the speechless patient is frequently reduced to the use of the most general propositions "yes" or "no," or both; that he may be unable to say "no" when told, although he says it readily in reply to questions requiring dissent; that he may be able ordinarily to put out his tongue well, as for example to catch a stray crumb, and yet unable to put it out when he tries, after being asked to do so; that he loses intellectual language and not emotional language; that although he does not speak, he understands what we say to him; and many other facts of the same order illustrate exactly the same principles as do such facts from other cases of disease of the nervous system as that in hemiplegia the arm suffers more than the leg; that most convulsions beginning unilaterally begin in the index-finger and thumb; that in cases of post-epileptic insanity there are degrees of temporary reduction from the least towards the most "organized actions," degrees proportional to the severity of the discharge in the paroxysm, or rather to the amount of exhaustion of the highest centres produced by the discharge causing the paroxysm. In all these cases, except in the instance of convulsion, which, however, illustrates the principle in another way, there are, negatively, degrees of loss of the most voluntary processes with, positively, conservation of the next most voluntary or next more automatic; otherwise put, there are degrees of loss of the latest acquirements with conservation of the earlier, especially of the inherited acquirements. Speaking of the physical side, there are degrees of loss of function of the least organized nervous arrangements with conservation of function of the more organized. There is in each reduction to a more automatic condition; in each there is dissolution, using this term as Spencer does, as the opposite of evolution.[1]

In *defects* of speech we may find that the patient utters instead of the word intended a word of the same class in meaning, as "worm-powder" for "cough-medicine," or in sound, as "parasol" for "castor oil." The presumption is that the patient uses what is to him a more "organized" or "earlier" word, and if so, dissolution is again seen. But often there is no obvious relation of any sort betwixt the word said and the one appropriate, and thus the mistake does not appear to come under dissolution. If, however, we apply the broad principles which we can, I think, establish from other cases of dissolution—viz., from degrees of insanity, especially the slight degrees of the post-epileptic insanity just spoken of—we shall be able to show that many of the apparently random mistakes in words are not real exceptions to the principle of dissolution.

For the above reasons I shall make frequent references to other classes of nervous disease. The subject is already complex without these excursions, but we must face the complexity. Dr. Curnow has well said (*Medical Times and Gazette*, November 29, 1873, p. 616), "The tendency to appear exact by disregarding the complexity of the factors is the old failing in our medical history."

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[1] Here I must acknowledge my great indebtedness to Spencer. The facts stated in the text seem to me to be illustrations from actual cases of disease, [illegible] conclusions he has arrived at deductively in his "Psychology." It is not affirmed that we have the exact opposite of evolution from the apparently brutal doings of disease; the proper opposite is seen in healthy senescence, as Spencer has shown. But from diseases there is, in general, the corresponding opposite of evolution.

Certain provisional divisions of our subject must be made. The reader is asked to bear in mind that these are admittedly arbitrary; they are not put forward as scientific distinctions. Divisions[1] and arrangements are easy. Distinctions and classifications are difficult. But in the study of a very complex matter we must first divide and then distinguish. This is not contradictory to what was said before on the necessity of encountering the full complexity of our subject. Harm comes, not from dividing and arranging, but from stopping in this stage, from taking provisional divisions to be real distinctions, and putting forward elaborate arrangements, with divisions and subdivisions, as being classifications. In other words we shall, to start with, consider our subject empirically, and afterwards scientifically. We first arbitrarily divide and arrange for convenience of obtaining the main facts which particular cases supply, and then try to classify the facts, in order to show their true relations one to another, and consider them on the psychical side as defects of mind, and on the physical side as defects of the nervous system. Empirically we consider the cases of affection of speech we meet with, as they *approach* certain nosological types (most frequently occurring cases); scientifically we classify the facts thus obtained, to show how affections of speech are *departures from* what we know of healthy states of mind and body. The latter study is of the cases as they show different degrees of nervous dissolution.

Let us first of all make a very rough popular division. When a person "talks" there are three things going on, speech, articulation, and voice. Disease can separate them. Thus from disease of the larynx, or from paralysis of its nerves, we have loss of voice, but articulation and speech remain good. Again, in complete paralysis of the tongue, lips, and palate, articulation is lost, but speech is not even impaired; the patient remains able to express himself in writing, which shows that he retains speech—internal speech—that he propositionizes well. Lastly, in extensive disease in a certain region in one half of the brain (left half usually) there is loss of speech, internal and external, but the articulatory muscles move well.

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[1] "How often would controversies be sweetened were people to remember that 'distinctions and divisions are very different things,' and that 'one of them is the most necessary and conducive to true knowledge that can be; the other, *when made too much of*, serves only to puzzle and confuse the understanding.' Locke's words are the germ of that wise aphorism of Coleridge: 'It is a dull or obtuse mind that must divide in order to distinguish; but it is a still worse that distinguishes in order to divide.' And if we cast our eyes back over time, it is the same spirit as that which led Anaxagoras to say, 'Things in this one connected world are not cut off from one another as if with a hatchet.'" [*Westminster Review* (art Locke), January, 1877 (no italics in original).]

Let us make a wider division. Using the term "language" we make two divisions of it, intellectual and emotional. The patient, whom we call speechless (he is also defective in pantomime), has lost intellectual language and has not lost emotional language.

The kind of case we shall consider first is that of a man who has lost speech and whose pantomime is impaired, but whose articulatory muscles move well, whose vocal organs are sound, and whose emotional manifestations are unaffected. This is the kind of case to be spoken of as No. 2 (p. 116).

The term "aphasia" has been given to affections of speech by Trousseau; it is used for defects as well as for loss of speech. I think the expression affections of speech (including defects and loss) is preferable. Neither term is very good, for there is, at least in many cases, more than loss of *speech*; pantomime is impaired; there is often a loss or defect in symbolizing relations of things in any way. Dr. Hamilton proposes the term "asemasia," which seems a good one. He derives it from "ἀ, and, *σημαίνω*, an inability to indicate by signs or language." It is too late, I fear, to displace the word aphasia. Aphasia will be sometimes used as synonymous with affections of speech in [illegible] article.

We must at once say briefly what we mean by speech [illegible] to what has been said by implication when excluding [illegible] this is popularly understood, and voice. To speak [illegible] words, it is to propositionize. A proposition is s[illegible] that it makes one new meaning; not by a [illegible] we call the separate meanings of the several [illegible] proposition are modified by each other. Single [illegible] and so is any unrelated succession of words. The [illegible] proposition. A single word is, or is in effect, a [illegible] words in relation are implied. The English tour[illegible] *d'hôte* was understood by the waiter to be askin[illegible] neighbours thought he was crying "oh" from [illegible] the use of a word that we judge of its proposit[illegible] "yes" and "no" are propositions, but only wh[illegible] dissent; they are used by healthy people interjec[illegible] propositionally. A speechless patient may retain the word "no," and yet have only the interjectional or emotional, not the propositional use of it; he utters it in various tones as signs of feeling only. He may have a propositional use of it, but yet a use of it short of that healthy people have, being able to reply "no" but not to say "no" when told; a speechless patient may have the full use of it. On the other hand, elaborate oaths, in spite of their propositional structure, are not propositions, for they have not, either in the mind of the utterer or in that of the person to whom they are uttered, any meaning at all; they may be called "dead propositions." The speechless patient may occasionally swear. Indeed he may have a recurring utterance, e.g. "Come on to me," which is propositional in structure but not, to him, propositional in use; he utters it on any occasion, or rather on no *occasion*, but every time he tries to speak.

Loss of speech is, therefore, the loss of power to propositionize. It is not only loss of power to propositionize aloud (to talk), but to propositionize either internally or externally, and it may exist when the patient remains able to utter some few words. We do not mean by using the popular term "power" that the speechless man has lost any "faculty" of speech or propositionizing; he has lost those words which serve in speech, the nervous arrangements for them being destroyed. There is no "faculty" or "power" of speech apart from words revived or revivable in propositions, any more than there is a "faculty" of [illegible]ation of movements apart from movements represented in [illegible] ways. We must here say, too, that besides the use of words [illegible] is a service of words which is not speech; hence we do [illegible]ession that the speechless man has lost words, but [illegible] words which serve in speech. In brief, speech[illegible] entire wordlessness.

[illegible] again that speech and words are psychical [illegible] course, anatomical substrata or bases as [illegible]ve. We must as carefully distinguish betwixt [illegible]sical bases as we do betwixt colour and its [illegible]hical state is always accompanied by a physical [illegible] the two things have distinct natures. Hence [illegible] the "memory of words" is a *function* of any [illegible]em, for function is a physiological term (*vide* [illegible]ther psychical state arises *during* not *from*— [illegible]inuity of a psychical state with a physical [illegible] nervous arrangements, which functioning is a purely physical thing—a discharge of nervous elements representing some impressions and movements. Hence it is not to be inferred from the rough division we have just made of the elements of "talking," and from what is said of their "separation" by disease, that there is anything in common even for reasonable contrast, much less for comparison, betwixt loss of speech (psychical loss) and immobiilty of the articulatory muscles from, say disease of the medulla oblongata, as in "bulbar paralysis" (a physical loss). As before said, we must not classify on a mixed method of anatomy, physiology, and psychology, any more than we should classify plants on a mixed natural and empirical method, as exogens, kitchen-herbs, graminaceæ, and shrubs. The things comparable and contrastable in a rough division are (1) the two physical losses: (*a*) loss of function of certain nervous arrangements in the cerebrum, which are not speech (words used in speech), but the anatomical substrata of speech and (*b*) loss of function of nervous arrangements in the medulla oblongata. (2) The comparison, on the psychical side, fails. There is no psychical loss in disease of the medulla oblongata to compare with loss of words, as this part of the nervous system, at least as most suppose,[1] has no psychical side; there is nothing psychical to be lost when nervous arrangements in the medulla oblongata are destroyed.

The affections of speech met with are very different in degree and kind, for the simple reason that the exact position of disease in the brain and its gravity differ in different cases; different amounts of nervous arrangements in different positions are destroyed with different rapidity in different persons. There is, then, no single well-defined "entity"—loss of speech or aphasia—and thus, to state the matter for a particular practical purpose, such a question as, "Can an aphasic make a will?" cannot be answered any more than the question, "Will a piece of string reach across this room?" can be answered. The question should be, "Can this or that aphasic person make a will?" Indeed, we have to consider degrees of affection of language, of which speech is but a part. Admitting the occurrence of numerous degrees of affection of language, we must make arbitrary divisions for the first part of our inquiry, which is an empirical one.

Let us divide roughly into three degrees: (1) *Defect of speech.*—

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[1] I, however, believe, as Lewes does, that in so far as we are physically alive we are psychically alive; that some psychical state attends every condition of activity of every part of the organism. This is, at any rate, a convenient hypothesis in the study of diseases of the nervous system.

The patient has a full vocabulary, but makes mistakes in words, as saying "orange" for "onion," "chair" for "table"; or he used approximative or quasi-metaphorical expressions, as "Light the fire up there" for "Light the gas." "When the warm water comes, the weather will go away," for "When the sun comes out, the fog will go away." (2) *Loss of speech.*—The patient is practically speechless, and his pantomime is impaired. (3) *Loss of language.*—Besides being speechless, he has altogether lost pantomime, and emotional language is deeply involved.

To start with, we take the simplest case, one of *loss of speech*, No. 2 ("complete aphasia"). Cases of defect of speech (1) are far too difficult to begin with, and so, too, are those cases (3) in which there is not only loss of speech, but also deep involvement of the least special part of language which we call emotional language. Moreover, we shall deal with a case of permanent speechlessness. I admit that making but three degrees of affection of language, and taking for consideration one kind of frequently occurring case, is an entirely arbitrary proceeding, since there actually occur very numerous degrees of affection of language, many slighter than, and some severer than, that degree (No. 2) we here call one of loss of speech. But, as aforesaid, we must study subjects so complex as this empirically before we study them scientifically; and for the former kind of study we must have what are called "definitions" by type, and state exceptions. This is the plan adopted in every work on the practice of medicine with regard to all diseases. Let us give an example of the twofold study: Empirically or clinically, that is for the art of medicine, we should consider particular cases of epilepsy, as each *approaches this or that nosological type* ("le petit mal, le grand mal," &c.). For the science of medicine we should, so far as is possible, consider cases of epilepsy, as each is dependent on a "discharging lesion" of this or that part of the cortex cerebri, and thus as it is a *departure from healthy states* of this or that part of the organism. We cannot do the latter fully yet, but the anatomico-physiological researches of Hitzig and Ferrier have marvellously helped us in this way of studying epilepsies, as also have the clinical researches of Broadbent, Charcot, Duret, Carville, and others.[1]

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[1] See Moxon, "On the Necessity for a Clinical Nomenclature of Disease," *Guy's Hospital Reports*, 1870, vol. xv, p. 479. In this paper Moxon shews conclusively the necessity of keeping the clinical, or what is above called empirical—not using that term in its popular bad signification—and scientific studies of disease distinct. After reading this paper, my eyes were opened to the confusion which results from mixing the two kinds of study. It is particu-

The following are brief and dogmatic statements about a condition which is a common one—the kind of one we call loss of speech, our second degree (No. 2) of affection of language. The statements are about two equally important things: (1) of what the patient has lost in language—his negative condition—and (2) of what he retains of language—his positive condition. Here, again, is an illustration of a general principle which is exemplified in many, if not in all, cases of nervous disease, and one of extreme importance, when they are scientifically considered as instances of nervous dissolution. We have already stated the duality of many symptomatic conditions in the remarks on p. 110. Without recognizing the two elements in all cases of affections of speech, we shall not be able to classify affections of speech methodically. If we do not recognize the duplex (negative and positive) condition, we cannot possibly trace a relation betwixt Nos. 1, 2, and 3 (p. 115). There can be no basis for comparison betwixt the wrong utterances in No. 1 and the non-utterances in Nos. 2 and 3—betwixt a positive and a negative condition—betwixt speech, however bad, and no speech. There is a negative and a positive condition in each degree; the comparison is of the three degrees of the negative element and the three degrees of the positive element; the negative and positive elements vary inversely. The condition of the patient No. 1, who made such mistakes as saying "chair" for "table" was duplex; (*a*) negatively in not saying "table," and (*b*) positively, in saying "chair" instead; there is in such a case *loss* of some speech, with *retention* of the rest of speech. Hence the term "defect of speech" applied to such a case is equivocal; it is often used as if the actual utterance was the *direct* result of the disease. The utterance is wrong in that the words of it do not fit the things intended to be indicated; but it is the best speech under the circumstances, and is owing to activity of healthy (except, perhaps, slightly unstable) nervous elements. The real, the primary, fault is in the nervous elements which do not act, which are destroyed, or are for the time *hors de combat*. If, then,

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larly important to have both an empirical arrangement and a scientific classification of cases of insanity. An example of the former is the much criticized arrangement of Skæ; the scientific classification of cases of insanity, like that of affections of speech, would be regarding them as instances of dissolution; the dissolution in insanity begins in the highest and most complex of all cerebral nervous arrangements, the dissolution causing affections of speech in a lower series. The one kind of classification is for diagnosis (for direct "practical purposes"), the other is for increase of knowledge, and is worthless for immediate practical purposes. The fault of some classifications of insanity is that they are mixed, partly empirical and partly scientific.

we compare No. 1 with No. 2, we compare the two negative conditions, the inability to say "table," &c. (the loss of some speech), in No. 1, with the loss of nearly all speech in No. 2, saying the latter is a greater degree of the former, and we compare the two positive conditions, the retention of inferior speech (the wrong utterances) in No. 1, with in No. 2 the retention of certain recurring utterances, and with the retention of emotional language, saying the latter is a minor or lower degree of language than the former. Unless we take note of the duplex condition in imperception (delirium and ordinary insanity) we shall not be able to trace a correspondence betwixt it and other nervous diseases. There are necessarily the two opposite conditions in all degrees of mental affections, from the slightest "confusion of thought" to dementia, unless the dementia be total.

THE PATIENT'S NEGATIVE CONDITION.

(1) *He does not speak.*—He can, the rule is, utter some jargon, or some word, or some phrase. With rare exceptions, the utterance continues the same in the same patient: we call these recurring utterances. The exceptions to the statement that he is speechless are two. (*a*) The recurring utterance may be "yes" or "no," or both. These words are propositions when used for assent or dissent, and they are so used by some patients who are for the rest entirely speechless. (*b*) There are occasional utterances. Under excitement the patient may swear; this is not speech, and is not exceptional; the oath means nothing; the patient cannot repeat it, he cannot *say* what he has just *uttered*. Sometimes, however, a patient, ordinarily speechless, may get out a phrase appropriate to some simple circumstance, such as "good-bye" when a friend is leaving. This is an exception, but yet only a partial exception; the utterance is not of high speech value;[1] he cannot *say* it again, cannot repeat it when entreated; it is inferior speech, little higher in value than swearing. However, sometimes a patient, ordinarily

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[1] What is meant by an utterance of high speech value, and by inferior speech, will later on be stated more fully than has been just now stated by implication. When we cease dealing with our subject empirically and treat it scientifically, we hope to show that these so-called exceptions come in place under the principle of dissolution. We may now say that speech of high value, or superior speech, is new speech, not necessarily new words and possibly not new combinations of words; propositions symbolizing relations of images new to the speaker, as in carefully describing something novel. It is the *latest* propositionizing. By inferior speech is meant utterances like, "Very well," "I do not think so," ready fitted to very simple and common circumstances, the nervous arrangements for them being well organized.

speechless, may get out an utterance of high speech value; this is very rare indeed.

(2) *He cannot write.*—That is to say, he cannot express himself in writing. This is called agraphia (William Ogle). It is, I think, only evidence of the loss of speech, and might have been mentioned in the last paragraph. Written words are symbols of symbols. Since he cannot write, we see that the patient is speechless, not only in the popular sense of being unable to talk, but altogether so; he cannot speak internally. There is no fundamental difference betwixt external and internal speech; each is propositionizing. If I say "gold is yellow" to myself, or think it, the proposition is the same; the same symbols referring to the same images in the same relation as when I say it aloud. There is a difference, but it is one of degree; psychically "faint" and "vivid," physically "slight" and "strong" nervous discharges. The speechless patient does not write because he has no propositions to write. The speechless man may write in the sense of penmanship; in most cases he can copy writing, and can usually copy print into writing, and very frequently he can sign his name without copy. Moreover, he may write in a fashion without copy, making, or we may say drawing, a meaningless succession of letters, very often significantly the simplest letters, pothooks. His handwriting may be a very bad scrawl, for he may have to write with his left hand. His inability to write, in the sense of expressing himself, is loss of speech; his ability to make ("to draw") letters, as in copying, &c., shows that his "image series" (the materials of his perception) is not damaged.

Theoretically there is no reason why he should not write music without copy, supposing, of course, that he could have done that when well; the marks (artificial images) used in noting music, have no relation to words any way used. On this matter I have no observations. Trousseau writes in his lecture on Aphasia (*New Sydenham Society's Transactions*, vol. i, p. 270), "Dr. Lasegue knew a musician who was completely aphasic, and who could neither read nor write, and yet could note down a musical phrase sung in his presence."

(3) In most cases the speechless patient *cannot read at all*, obviously not aloud, but not to himself either, including what he has himself copied. We suppose our patient cannot read. This is not from lack of sight, nor is it from want of perception; his perceptio[illegible]t, itself in fault, as we shall see shortly.

(4) His power of making signs is impaired (pantomimic propositionizing). We must most carefully distinguish pantomime from gesticulation. Throwing up the arms to signify "higher up," pantomime, differs from throwing the arms when surprised, gesticulation, as a proposition does from an oath.

So far we have, I think, only got two things, loss of speech (by simple direct evidence, and by the indirect evidence of non-writing and non-reading) and defect of pantomime. There are in some cases of loss of speech other inabilities: the most significant are that a patient cannot put out his tongue when he tries, or execute other movements he is told, when he can move the parts concerned in other ways quite well.

THE PATIENT'S POSITIVE CONDITION.

(1) He can understand what we say or read to him; he remembers tales read to him. This is important, for it proves that, although speechless, the patient is not wordless. The hypothesis is that words are in duplicate; and that the nervous arrangements for words used in speech lie chiefly in the left half of the brain; that the nervous arrangements for words used in understanding speech (and in other ways) lie in the right also. Hence our reason for having used such expressions as "words serving in speech"; for there is, we now see, another way in which they serve. When from disease in the left half of the brain speech is lost altogether, the patient understands all we say to him, at least on matters simple to him. Further, it is supposed that another use of the words which remain is the chief part of that service of words which in health precedes speech; there being an unconscious or subconscious revival of words in relation before that second revival which is speech. Coining a word, we may say that the process of verbalizing is dual; the second "half" of it being speech. It is supposed also that there is an unconscious or subconscious revival of relations of images, before that revival of images in relation which is perception.

(2) His articulatory organs move apparently well in eating, drinking, swallowing, and also in such utterances as remain always possible to him (recurring utterances), or in those which come out occasionally. Hence his speechlessness is not owing to disease of those centres in the medulla oblongata for immediately moving the articulatory muscles; for in other cases of nervous disease, when these centres are so damaged [illegible] the articulatory muscles are so much paralysed that *talking* is [illegible]ble, the patient remains able to *speak* (to propositionize) as well [illegible] he has internal speech, and can write what he speaks.

The following dicta may be of use to beginners. Using the popular expression "talk," we may say that if a patient does not talk because his brain is diseased, he cannot write (express himself in writing), and can swallow well; if he cannot talk because his tongue, lips, and palate are immovable, he can write well and cannot swallow well.

(3) His vocal organs act apparently well; he may be able to sing.

(4) His emotional language is apparently unaffected. He smiles, laughs, frowns, and varies his voice properly. His recurring utterance comes out now in one tone and now in another, according as he is vexed, glad, &c.; strictly, we should say he sings his recurring utterance; variations of voice being rudimentary song (Spencer); he may be able to sing in the ordinary meaning of that term. As stated already, he may swear when excited, or get out more innocent interjections, simple or compound (acquired parts of emotional language). Although he may be unable to make any but the simplest signs, he gesticulates apparently as well as ever, and probably he does so more frequently and more copiously than he used to do. His gesticulation draws attention to his needing something, and his friends guess what it is. His friends often erroneously report their guessing what he wants when his emotional manifestations show that he is needing something, as his expressing what thing it is that he wants.

So far for the negative and positive conditions of language in our type case of loss of speech—No. 2 in defect of language.

Words are in themselves meaningless, they are only symbols of things or of "images" of things; they may be said to have meaning "behind them." A proposition symbolizes a particular relation of some images.[1]

We must, then, briefly consider the patient's condition in regard to the images symbolized by words. For although we artificially separate speech and perception, words and images co-operate intimately in most mentation. Moreover, there is a morbid condition in the image series (imperception), which corresponds to aphasia in the word series. The two should be studied in relation.

The speechless patient's perception (or "recognition," or "thinking" of things) (propositions of images) is unaffected, at any rate as regards simple matters. To give examples: He will point to any object he knew before his illness which we name; he recognizes drawings of all objects he knew before his illness. He continues able to play at cards or dominoes; he recognizes handwriting, although he cannot read the words written; he knows poetry from prose, by the different endings of the lines on the right side of the page. One of my patients found out the continuation of a series of papers in a magazine volume, and had the right page ready for her husband when he returned from his work; yet she, since her illness, could not read a word herself, nor point to a letter nor could she point to a figure on the clock. There is better and simpler evidence than that just adduced that the image series is unaffected; the foregoing is intended to show that the inability to read is not due to loss of perception nor to non-recognition of letters, &c., as particular marks or drawings, but to loss of speech. Written or printed words cease to be symbols of words used in speech for the simple reason that those words no longer exist to be symbolized; the written or printed words are left as symbols of nothing, as mere odd drawings. The simplest example showing the image series to be undamaged is that the patient finds his way about; this requires preconception, that is, "propositions of images" of streets, &c. Moreover, the patient can, if he retains the propositional use of "yes" and "no," or if he has the equivalent pantomimic symbols, intelligently assent or dissent to simple statements, as that "racehorses are the swiftest horses," showing that he retains organized nervous arrangements for the images of the things "swiftness" and "horse"; this has already been implied when it was asserted that he understands what we say to him, a process requiring not some of his words only, but also some of his "images" of things, of which the words are but symbols.

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[1] The term "image" is used in a psychical sense, as the term "word" is. It does not mean "visual" images only, but covers all mental states which represent things Thus we speak of auditory images. I believe this is the way in which Taine uses the term "image." What is here called "an image" is sometimes spoken of as "a perception." In this article the term "perception" is used for a *process*, for a "proposition of images," as speech is used for propositions, i.e., particular inter-relations of words. The expression "organized image" is used briefly for "image," the *nervous arrangements for which* are "organized" correspondingly for "organized word," &c.

Such facts as the above are sometimes adduced as showing that the patient's "memory" is unaffected. That expression is misleading, if it implies that there is a general faculty of memory. There is no faculty of memory apart from things being remembered; apart from having, that is, now and again, these or those words, or images, or actions (faintly or vividly). We may say he has not lost the memory of images, or better, that he has the images actually or potentially, the nervous arrangements being intact and capable of excitation did stimuli come to them; we may say that he has lost the memory of those words which serve in speech. It is better, however, to use the simple expression that he has not lost images, and that he has lost the words used in speech.

These facts as to retention of images are important as regards the writing of speechless patients. The printed or written letters and words are images, but they differ from the images of objects, in being artificial and arbitrary, in being acquired later; they are acquired after speech and have their meaning only through speech; written words are symbols of images. The aphasic patient cannot express himself in writing because he cannot speak; but the nervous arrangements for those arbitrary images which are named letters are intact, and thus he can reproduce them as mere drawings, as he can other images, although with more difficulty, they, besides lacking their accustomed stimulus, being less organized. He can copy writing, and he can copy print into writing. When he copies print into writing, obviously he derives the images of letters from his own mind (physically his own organization). He does not write in the sense of expressing himself, because there are no words reproduced in speech to express. That series of artificial images which make up the signature of one's name has become almost as fully organized as many ordinary images; hence in many cases the speechless man who can write nothing else without copy can sign his name.

For the perception (or recognition or thinking) of things, at least in simple relations, speech is not necessary, for such thought remains to the speechless man. Words are required for thinking, for most of our thinking at least, but the speechless man is not wordless; there is an automatic and unconscious[1] or subconscious service of words.

It is not, of course, said that speech is not required for thinking on novel and complex subjects, for ordinary images in new and complex relations (i.e., to the person concerned), and thus the process of perception in the speechless, but not wordless, man may be defective in the sense of being inferior from lack of co-operation of speech; it is not itself in fault, it is left unaided.

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[1] The expression "unconscious reproduction of words," involves the same contradiction as does the expression, "unconscious sensation." Such expressions may be taken to mean that energizing of lower, more organized, nervous arrangements, although unattended by any sort of conscious state, is essential for, and leads to, particular energizings of the highest and least organized—the now organizing nervous arrangements, which last-mentioned energizing is attended by consciousness I, however, think (as Lewes does) that some consciousness or "sensibility" attends energizing of all nervous arrangements (I use the term "subconscious" for slight consciousness). In cases where from disease the highest nervous arrangements are suddenly placed *hors de combat*, as in sudden delirium, the next lower spring into greater activity, and then, what in health was a subordinate subconsciousness, becomes a vivid consciousness, and is also the highest consciousness there then can be.

To understand anything novel and complex said to him, the healthy man speaks it to himself, e.g., repeats, often aloud, complex directions of route given to him.

The word "thing" has not been used as merely synonymous with "substance"; nor is it meant that anybody has nervous arrangements for the images of "swiftness" and "horse," but only for images of some swiftly moving thing or things, and for images of some particular horse or horses.

It may be well here to give a brief recapitulation of some parts of our subject and, also very briefly, an anticipation of what is to come; the latter is given partly as an excuse for having dwelt in the foregoing on some points not commonly considered in such an inquiry as this, and partly to render clearer some matters which were only incidentally referred to.

The division into internal and external speech (see p. 119) is not that just made into the dual service of words. Internal and external speech differ in degree only. Such a difference is insignificant in comparison with that betwixt the prior unconscious, or subconscious, and automatic reproduction of words and the sequent conscious and voluntary reproduction of words; the latter alone is speech, either internal or external. Whether I can show that there is this kind of duality or not, it remains certain that our patient retains a service of words, and yet ordinarily uses none in speech. The retention of that service of words which is not a speech use of words, is sometimes spoken of as a retention of "memory of" words, or of "ideas of" words. But as there is no memory or idea of words apart from having words, actually or potentially, it is better to say that the patient retains words serving in other ways than in speech; we should say of his speechlessness, not that he has lost the memory of words, but simply that he has lost those words which serve in speech.

When we consider more fully the duality of the verbalizing process, of which the second "half" is speech, we shall try to show that there is a duality also in the revival of the images symbolized; that perception is the termination of a stage beginning by the unconscious or subconscious revival of images which are in effect "image symbols"; that we think not only by aid of these symbols, ordinarily so-called (words), but by aid of symbol-images. It is, I think, because speech and perception are preceded by an unconscious or subconscious reproduction of words and images, that we seem to have "faculties" of speech and of perception, as it were, above and independent of the rest of ourselves.

We seem to have a memory or ideas [1] of words *and* words; having really the two kinds of service of words. The evidence of disease shows, it is supposed, that the highest mentation arises out of our whole organized states, out of ourselves, that will, memory, &c., "come from below," and do not stand autocratically "above," governing the mind; they are simply the now highest, or latest, state of our whole selves. In simple cases of delirium (partial imperception with inferior perception) as when a patient takes his nurse to be his wife, we find, I think, a going down to and a revelation of what would have been when he was sane, the lower and earlier step towards his true recognition or perception of the nurse.

The first step towards his recognition of her when he was sane would be the unconscious or subconscious, and automatic reproduction of his, or of one of his, well-organized symbol-images of woman; the one most or much organized in him would be his wife. To say what a thing is, is to say what it is like; he would not have known the nurse even as a woman, unless he had already an organized image of at least one woman. The popular notion is, that by a sort of faculty of perception, he would recognize her without a prior stage in which, he being passive, an organized image was roused in him by the mere presence of the nurse; the popular notion almost seems to imply the contradiction that he first sees her, in the sense of recognizing her, and then sees her as like his already acquired or organized image of some woman. We seem to ourselves to perceive, as also to will and to remember, without prior stages, because these prior stages are unconscious or subconscious. It seems to me that in delirium the patient is reduced to conditions which are revelations of, or of parts of, the lower earlier and prior stages; the lower or earlier stages are then conscious. They are the *then* highest or *latest* conscious states. When the patient becomes delirious, he takes the nurse to be his wife. More or fewer of the highest nervous arrangements being then exhausted, the final stage is not possible. There is only the first stage; the reproduction of his well-organized symbol-image is all there is, and that is all the nurse can be to him; she is, to him, his wife. The symbol-image is then vividly reproduced because the centres next lower than those exhausted are in abnormally great activity (note that there are two conditions, one negative and the other positive). There is a deepening of consciousness in the sense of going down to lower earlier and more organized states, which in health are mostly unconscious or subconscious, and precede higher or later conscious states; in other words, with loss with or defect of object consciousness, even in sleep dreaming, there is increasing subject consciousness; on the physical side, increasing energizing of those lower centres which are in the daytime more slightly energizing during that unbroken subconscious "dreaming," from which the serial states, constituting our latest or highest object consciousness, are the continual "awakenings."

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[1] The so-called *idea* of a word, in contradistinction to *the* word, is itself a word subconsciously revived, or revivable, before the conscious revival or revivability of the same word, which latter, in contradistinction to the so-called *idea* of a word, is the so-called *word itself*—*the* word.

It is supposed that the well-organized images spoken of, in effect arbitrary images, symbol-images, those which *become* vivid and are "uppermost" in delirium, and then cease to be mere symbols, constitute what seems to be a "general notion" or "abstract idea" of such things as "horse," "swiftness," &c.; their particularity (that they are only images of some horse or horses, of some swift moving thing or things) not appearing, because they are unconscious or subconscious; they served once as images of particular things, and at length as symbol-images of a class of images of things, as well as images of the particular things.

At p. 120 we spoke of the right half of the brain as being the part during the activity of which the most nearly unconscious and most automatic service of words begins, and of the left as the half during activity of which there is that sequent verbal action which is speech. The division is too abrupt; some speech—voluntary use of words—is, as we have seen when alluding to occasional utterances, possible to the man who is rendered practically speechless by disease in the left half. Again, from disease of the right half, there is not loss of that most automatic service of words which enables us to understand speech. The thing which it is important to show is, that mentation is dual, and that physically the unit of function of the nervous system is double the unit of composition; not that one-half of the brain is "automatic" and the other "voluntary."

Having now spoken of the kind of case we shall consider, and having added remarks, with the endeavour to show how the several symptoms—negative and positive—are related one to another, we shall be able to give reasons for excluding other kinds of cases of speechlessness.

We are not concerned with cases of all persons who do not speak. We shall not, for example, deal with those untrained deaf-mutes who never had speech, but with the cases of those persons only who have had it, and lost it by disease. The condition of an untrained deaf-mute is in very little comparable with that of our arbitrarily taken case of loss of speech. The deaf-mute's brain is not diseased, but, because he is deaf, it is uneducated (or in anatomical and physiological phraseology undeveloped) so as to serve in speech. Our speechless patient is not deaf. Part of our speechless patient's brain is destroyed; he has *lost* nervous arrangements which had been trained in speech. Moreover, our speechless man retains a service of words which is not speech; untrained deaf-mutes have no words at all. Further, the untrained deaf-mute has his natural system of signs, which to him is of speech value as far as it goes. He will think by aid of these symbols as we do by aid of words.[1] Our speechless patient is defective even in such slight pantomime as we may reasonably suppose to have been easy to him before his illness. The deaf-mute may have acquired for talking and thinking the common arbitrary system of deaf-mute signs (finger-talk), or he may have been taught by the new method to speak as we do, and thus have ceased to be mute. But when not taught to speak, he is not in a condition even roughly comparable with that of a man who has *lost* speech. No doubt by disease of some part of his brain the deaf-mute might lose his natural system of signs, which are of some speech value to him, but he could not lose speech, having never had it. Much more like our speechless patient's condition is that of the little child which has been taught to understand speech, and has not yet spoken.

There is another set of cases of so-called loss of speech, which we shall not consider as real loss of speech. I prefer to say that these patients *do* not speak: cases of some persons are meant, who do not talk and yet write perfectly. This may seem to be an arbitrary exclusion. There is in most of these cases an association of symptoms, which never arises from any local disease of any part of the nervous system; the so-called association is a mere jumble of symptoms. Let us state the facts. The patients are nearly always boys or unmarried women. The bearing of this is obvious. The so-called loss of speech is a total non-utterance, whereas it is an excessively rare thing for a patient who does not speak, because his brain is locally diseased, to have no utterance whatever; I do not remember seeing one such case in which there was not some utterance (recurring utterance) a few days or a few weeks after the onset of the illness; the absolute pseudo-speechlessness may remain for months. They cannot be mute from paralysis of the articulatory muscle, because they swallow well. Frequently there is loss of voice also—they get out no sounds except, perhaps, grunts, &c.—and yet they cough ringingly and breathe without hoarseness or stridor; there is no evidence of laryngeal disease. Now loss of voice never occurs with loss of speech from local disease of one side of the brain. No disease of the larynx would cause loss of speech or loss of articulation. The patients often "lose" their speech after calamity or worry. In these cases there is no hemiplegia and no other one-sided condition from first to last. They often, after months of not speaking, recover absolutely and immediately after some treatment which can have no therapeutical effect, e.g., a liniment rubbed on the back, a single faradaic stimulation of the vocal cords or of the neck. Dr. Wilks has reported a case of "cure" of a girl who had not spoken for months; she had also "lost" the use of her legs. Knowing well what was the general nature of the case, Dr. Wilks, by speaking kindly to her, and giving her an excuse for recovery in the application of faradization, got her well in a fortnight. Sometimes the so-called speechless patient speaks inadvertently when suddenly asked a question, and then goes on talking; is well again. Sometimes speech is surprised out of her. Thus a woman, whose case is recorded by Durham, when told to cry "Ah!" when the spatula was holding down her tongue, pushed his hand away, saying, "How can I, with that thing in my mouth?" She then said, "Oh! I have spoken." She was "cured." I believe that patients, "speechless" as described, might be "cured" by faradization of the vocal cords, or by a thunderstorm, or by quack medicines or appliances, or by mesmerism, or by wearing a charm, or —not speaking flippantly—by being "prayed over."

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[1] We must not confound the finger-talk with the "natural" system of signs. They are essentially different. No one supposes that words are essential for thought, but only that some symbols are essential for conceptual thought, although it may be that people with "natural" symbols do not reach that higher degree of abstract thinking which people do who have words.

Sometimes these cases are spoken of as cases of "emotional aphasia" —the speechlessness is said to be "caused by" emotional excitement, because it often comes on *after* emotional disturbance.

I submit that the facts that the patients do not talk and *do* write and *do* swallow are enough to show that there is no disease at all, in any sense except that the patients are hysterical (which is saying nothing explanatory); or that they are pretending. There can be no *local* disease, at any rate.

These cases are spoken of at length, although they are excluded, because they are sometimes adduced as instances of aphasia, or loss of speech proper, with ability to write remaining. I confess that were I brought face to face with a man whom I believed to *have* local disease of his brain, who did not *talk*, and yet wrote well, I should conclude that he did *speak* internally although he could not talk. To say that *he* cannot speak, and yet can express himself in writing is equivalent, I think, to saying *he* cannot speak and yet *he* can speak.

ON AFFECTIONS OF SPEECH FROM DISEASE OF THE BRAIN.

[BRAIN, 1880, vol. ii, p. 203.]

IN the first[1] instalment of this article it was pointed out that there are necessarily numerous degrees and kinds of affection of language, since "different amounts of nervous arrangements in different positions are destroyed with different rapidity in different persons." Moreover, cases are vastly different in their different stages; a patient may be quite speechless for a few days, and afterwards improve so as to have at length only slight defect of speech; and of course there are numerous cases of complete recovery. It is necessary to make some division of cases; we roughly made three groups. We were careful to declare that this division was an arbitrary one, that it was not a scientific distinction. The divisions usually made are arbitrary, too, although the nomenclature being in highly technical psychological and clinical terms, they may appear to the unwary as being real, almost natural, distinctions. As was then insisted on, we must in an empirical inquiry take type-cases; we follow the plan which is tacitly, if not avowedly, adopted in every work on the "Practice of Medicine" with regard to all diseases.

We took for first consideration the simplest group, cases of loss of speech (No. 2) p. 116; cases of defect of speech (No. 1), and that deeper involvement of language in which emotional manifestations (No. 3) scarcely remain, are more difficult and will be considered later. We take the simple case for investigation first, just as, were we writing on hemiplegia, we should take first the simplest case of that paralysis, not the more difficult case, in which there are deep loss of consciousness and lateral deviation of the eyes and head, as well as paralysis of the face, tongue, arm, and leg. A patient who loses speech may regain it; it is convenient to consider cases of permanent speechlessness.

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[1] I should like to remark that one very general conclusion to which the several facts so far stated, and facts afterwards to be stated, point, was in principle long ago formulated by M. Baillarger. So far back as 1866 [p. 53] I made the following quotations from his writings, which I now reproduce :—

"L'analyse des phénomènes conduit à reconnaître, dans certains cas de ce genre, que l'incitation verbale involontaire persiste, mais que l'incitation volontaire est abolie. Quant à la perversion de la faculté du langage caractérisée par la prononciation de mots incohérents, la lésion consiste encore dans la substitution de la parole automatique à l'incitation verbale volontaire."

I ought to have reproduced this quotation in the first instalment of this article, as evidently I am following pretty closely the principle this distinguished Frenchman has laid down. For the satisfaction of curious persons, I may say that I give it now spontaneously, no one having drawn my attention to the omission. I fear M. Baillarger's acute remarks have attracted little attention, and I say with regret that I had forgotten them. I do not remember from what book I took the quotation.

On pp. 116 *et seq.* we made some brief general statements as to the speechless patient's condition; we especially insisted on the necessity of recognizing a positive as well as a negative element. I feel convinced that unless in all degrees of affection of language we recognize that the symptomatic condition is duplex, we shall not trace relations betwixt them, and shall be misled into supposing that cases are different in kind when there are only differences of degree. We have remarked on this (p. 117). Further, we shall not be able to trace analogies betwixt these examples of dissolution beginning in the lower cerebral centres, and cases of dissolution beginning in the highest centres, that is to say, cases of insanity where the condition is manifestly duplex. The most important thing showing the duality of the speechless man's condition is given very generally by saying that speechlessness does not imply wordlessness. We stated that there is not evidence that the process of perception is damaged in itself; we say "in itself," admitting that perception may suffer from lack of co-operation of speech-use of words.

We have now to consider more particularly the condition of the patient we call speechless. In most cases there remains some utterance. But to utter words is not necessarily to speak. To speak is to propositionize. We admit, however, that in some cases which we call loss of speech there is a recurring utterance, viz., "yes" or "no," which is of propositional value; and that occasionally there occur utterances which are of propositional value, and are made up of several words. But in these cases the recurring utterances which have propositional value are so very general in their application, and the occasional utterances which have propositional value are so rare and usually also of so very general application, that it is almost pedantic to say the patients are not speechless. Nevertheless there are exceptions to our statement that there is *loss* of speech in the type case (No. 2) and full consideration will be given to them.

We divide the utterances into two classes, recurring and occasional.

(1) RECURRING UTTERANCES.

Soon after the attack there may be no sort of utterance. But almost always one comes in a few days or weeks. I used to call them "stock utterances." They are always utterable, and they alone remain with the exception of the rare occasional utterances. We make four divisions of recurring utterances.

(1) It is sometimes jargon. In one case it was "yabby," in another "watty." Sometimes there is a succession of different jargon; in one case, "Me, me committimy, pittymy, lor, deah." The patient utters his jargon at any time. If he *says* anything, it is always "yabby," or whatever his jargon may be; in reality he *says* nothing with these utterances; they have no propositional value whatever.

(2) Sometimes the utterance is, what to a healthy person is a word, as "man," "one," "awful," &c. Such a word is, for use, no better than jargon in the mouth of the speechless patient; it is not a word to him; "man" as a recurring utterance is not a symbol for a human being. The so-called word comes out, just as "yabby" does and means no more, means nothing. A single word might have, in a healthy person, propositional value. For example, were a person asked how many oranges he would buy, the reply "one" would be a proposition. But the speechless man's recurring "one" comes out whenever anything comes out, and applies to nothing at all.

Here, having given some examples, I may make one general remark about all kinds of recurring utterances. The rule is that the particular recurring utterance each person has at first never changes. Sometimes, however, but exceedingly rarely, it changes. A patient of mine, for some months when under my care in the London Hospital, could only utter the word "Dick." Later it changed to "Jimmy," with the variations of "Jin" and Jigger." Another general remark is that although these rags and tatters of what was once the patient's speech are of no use as speech, they serve as parts of emotional manifestations; it is rather, we should say, the tones in which they are uttered; it would be most correct to say the patient "sings" his recurring utterances—variations of tone with healthy speech being rudimentary singing (Spencer).

In this service of these as also in that of the other recurring utterances, we have evidence that emotional language is not affected.[1]

The way in which speechless recurring utterances serve patients is exemplified in some cases noted in the Hospital Reports of the *Lancet* [7], and [15]. Several other things of importance for other departments of our subject are given in the following extracts:—

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[1] I would not affirm that the finest emotional manifestations may not be lost in cases of loss of speech; I do not know that they are. It would be very remarkable if they were not.

"The patient we saw could only utter the word 'Dick' and this word he uttered whenever we asked him a question. We were told that when the man was vexed by the other patients in his ward he would swear. He generally used the common explosive sound so much in favour with English swearers. He could not, however, say the word when required to do so, even whilst it was well kept before his mind by frequent repetition. He seemed to make efforts to say it, but the word 'Dick' always came out instead. The oath was only uttered under the influence of emotion, and could never be repeated at will" (p. 43).

The following is a further note of the same case from the "Mirror" of the *Lancet*, July 20, 1867.

"When the poor fellow left the London Hospital he was able to utter the word 'Dick' only, except that he swore when vexed. He is now in a workhouse where Dr. Hughlings-Jackson saw him a few weeks ago. Strange to say, the patient's stock phrase is now 'Jimmy'; he never says 'Dick.' Although it is two years since the patient left the hospital, as soon as he saw the doctor he raised himself eagerly from his chair, offered his left hand—his right is still paralysed—and cried out very vivaciously, 'Jimmy, Jimmy,' &c., evidently pleased to see some one whom he knew. The ward superintendent says the patient sometimes sings; that the word he then uses is 'jigger.' He is usually quiet; but when vexed he swears, or rather utters a very nasty word, the last syllable of which rhymes to the last syllable of jigger.[1] He cannot say this word when he tries, but when trying, says 'Jim' instead. 'Jimmy' seems to be the word he uses as an ejaculation to show states of feeling, and 'Jim' when he is trying to convey information. When asked to show how many children he had, he extended his left five digits twice, and at each extension he uttered jerkingly the word 'Jim.' At a second visit, the man replied to the same question in the same way; but there are no means of knowing whether his reply is a correct one or not. He does not tell the number of days in the week by this plan. He sang when asked; and although the performance was of the very poorest kind, there was cadence with variation of tone. In one of these efforts he used as a vehicle of sound the word 'Jim,' in another 'jigger.' The ward superintendent remarked that the man's friends had not visited him since Christmas. Here the patient clenched his fist, tightened his lips, face and neck, holding his breath, and turning red the while as if making an effort. After a moment or two he sighed deeply and relaxed, shook his head, and looked as if he had given up an attempt to do something. The attendant believed the patient was 'trying to talk,' and said he often saw him put himself in that way. As Dr. Hughlings-Jackson was leaving the room, the patient left by another door, but in passing through the doorway he stopped, and turned his head as if he had suddenly remembered something, looked towards the doctor, and said pleasantly, 'Jim, Jim.' It was supposed that this meant good-bye."

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[1] "Bugger."

I may here mention that I did not get to know if the patient had sons of the name of Richard and James (*vide infra*).

The following is from the same "Mirror" of the *Lancet* as the last quotation. It shows a certain *use* of the jargon "ow," during the expression of number, as well as its use as the proposition "yes."

"In another workhouse Dr. Hughlings-Jackson saw a woman, aged 25, who is only able to utter the phrase 'Oh! my God!' and the noise 'ow,' probably a corruption of 'oh!' When the doctors went up to her bed and spoke to her, she cried out 'Oh! my God!' When next spoken to, she said 'Oh!' and then put her hand over her mouth. She uttered the phrase several times in the interview; but she 'spoke' with the syllable 'ow,' expressing assent or dissent by the tone she gave to it, and by her manner. She was asked how long it was after her confinement before the loss of speech came on. She held out her five left fingers, and said 'Ow,' and then separating one finger from the rest of those of the paralysed right hand, again said 'ow.' The doctors said interrogatively, 'Six?' She nodded, and said 'Ow.' They then asked whether weeks, months, or years, really themselves knowing the right time. By variation of tone of 'ow' by nodding and shaking the head, she expressed assent or dissent when the right or wrong period was named. She laughed heartily when something jocose was said, crying out "Oh! my God!' When the death of her baby was mentioned, her eyes filled with tears. The nurse says the woman was once in her ward before, and then the words uttered were 'Oh! my goodness will!'"

(3) The recurring utterance is sometimes a phrase. In one case "Come on," or sometimes that patient uttered "Come on to me." In another case, just mentioned, it was "Oh! my God!" In another case, mentioned to me by Dr. Langdon Down, "Yes, but you know."

In some cases, as in the one first mentioned, the patient may utter "yes" or "no," or both, in addition to his recurring phrase (see 4). He has then the two sets of recurring utterances.

These phrases, which have propositional structure, have in the mouths of speechless patients no propositional function. They are not speech, being never used as speech; they are for use only compound jargon: they or their tones are at the best of interjectional value only. The man who uttered "Come on to me," uttered it on every occasion when he made a rejoinder to anything said to him.

(4) A common thing is that the patient retains as his sole utterance "yes" or "no," or both these words. Sometimes there is, in addition, some utterance of one of the other divisions. This must be carefully borne in mind. We shall consider the utterances "yes" and "no" at length. It is the most important part of the whole inquiry. The consideration of these and of some other fundamentally like phenomena will help us out of the empirical stage of divisions into the scientific one of distinctions.

To speak is, as has been said, to propositionize; many verbal utterances by the healthy are not speech. Now the words "yes" and "no" are propositions; indeed, to call them "words" is not to acknowledge their proper rank; "proposition words" might be a more correct expression. But they are not always propositions, are not always used for assent and dissent; and thus the term "word" is convenient, if not strictly accurate.

It does not matter what the philological history of the words may be; at any rate "yes" and "no" stand for propositions. They are propositions in effect; we can *say* with them. Nor must we limit ourselves to the very syllables "yes" and "no." One of my patients had the utterance "Eh," which was "yes" for him, and possibly was a corruption of his healthy "yes." Were we now dealing with the less special part of intellectual language, pantomime, we should admit nodding the head for assent and shaking the head for dissent to be pantomimic propositions. With the other recurring utterances (1), (2) and (3), the patient says nothing; they are a mere series of syllables; the so-called words and phrases (2 and 3) being intellectually dead. Is not there then in the utterance of "yes" and "no" a real exception to the statement that our patient is speechless?

An utterance is or is n[illegible]position according as it is used.

(*a*) The speechless [illegible] utter "yes" or "no," or both, in different tones, merely [illegible] he is thus or thus excited. It is then not a proposition, but an interjection, a mere vehicle for variations of voice, expressive of feeling. (*b*) He may have this service of the words and be able also to reply with them; the latter is a propositional use of them. (*c*) He may, in addition to (*a*) and (*b*), be able to say the words when told to say them.

(*a*) A speechless patient may utter "yes" and "no" without any sort of application. He may utter "yes" when he means "no," and "no" when he means "yes." He may nod when he utters "yes." He may affirm or deny by the less special language of pantomime when he cannot *use* for affirmation and denial the words of affirmation and denial which he can glibly utter. They are not, therefore, propositions to him. Nevertheless, this low degree of the utterances serves him. He utters "Yes," "yes," "yes," or "No," "no," "no," merrily, or he utters them sadly, when respectively glad or sorry. That is to say, although he has not the propositional use of "yes" and "no," there is that emotional service of them which other speechless patients have of their recurring jargon, words or phrase (1, 2, 3). His utterances of them in various tones are revelations to *us* of his varying emotional states. We must be careful not to give *such* utterances of "yes" and "no" the credit of being propositions. From the tones in which they are uttered, *we* may understand or guess how the patient is feeling; and with the conspiring aid of the then circumstances, we or his friends may often *infer* what he is thinking. But so we could by the tones in which the recurring jargon (1) or phrase (2) is uttered. Like smiles, they are, when so used, not signs for emotional states, but they are, or rather the tones of them are, *parts of* this or that emotional manifestation. On the other hand, if the patient who generally uttered "no" at random, *used* "no" in a particular tone, in order to signify that he dissented, it would be speech, or at any rate of speech value; and so it would be speech or of speech value if a speechless man used his jargon with the same intention. The women who uttered "ow" could express assent or dissent by the different tone she gave to it (p. 134). The following is a striking case:—

I have seen a patient who nearly a year before had become rapidly apoplectic. On recovering from this condition, he uttered only "low," but soon he uttered "no," and when I saw him he had nearly recovered speech. His articulation was defective, but his wife could understand what he said, not merely guess his me[illegible]nd I could nearly always do the same. He could express hi[illegible]riting, and could read. For the moment using popular lang[illegible]*ut prejudice*"), he had the mental power of speech, but had defect in the executive. But he very often uttered the word "no" when he meant "yes"; this is a very rare thing in the midst of so much recovery of speech. In reply to one of my questions, he uttered "No, no." His wife said he meant "yes"; he nodded. Later in our investigation he uttered "no"; but his medical attendant, alive to his misuse of that word, said, "Do you mean 'no'?" The patient showed that he did by re-uttering it in a ceremonious, slow, decided tone. Thus the patient *uttered* in a tone to signify dissent, the word which, as usually uttered by him, would not have meant dissent.

Here plainly "no" was not a proposition, but the tone it was uttered in was of propositional value—at least vocal pantomime. It matters not what trick or dodge (tone of voice, cardsharper's smile, &c.), be used to express assent or dissent, or to express any relation betwixt things; if so used, there is a proposition.

Some years ago I had under my care in the London Hospital a man whose sole utterance was "no," and something like "eh," which was "yes" to him. His wife told me he could make the children "behave when they were at the top yard" by shouting out "No," "no," "no," in an angry tone. These emotional utterances of his may seem to have some slight propositional flavour; he may have *used* "no" in an angry tone, not merely uttered it during vexation.[1] This, however, is doubtful. The patient could reply "no," but the slight degree of his power of expressing himself may be judged of by his way of getting his children to understand what he wanted. He would make one stand before him; she would guess one thing after another until, by quickness or by lucky accident, she guessed what he wanted, or until he knocked her down with his fist. I shall have to refer to this case several times, and shall therefore call the patient "Dow."

(*b*) In some cases of loss of speech there is a use of the word "yes" and "no," which is higher than a mere emotional service. The patient can reply with them. Here, then, is evidence that the so-called speechless man is not absolutely speechless; he propositionizes by "yes" and "no." Now we have come to a very important matter. In the case of "no," at least the use of that word does not in all patients reach the level of normal speech; or, speaking more correctly, the patient cannot utter that word in all the ways healthy people can. He may be, as aforesaid, able to reply "no" to a question requiring dissent, when he cannot say the word when he is told and when he tries. This has been observed and commented on by Sir Thomas Watson, in the last edition of his "Practice of Physic." I found it out when giving a clinical demonstration of "Dow's" case. I told the students that he could utter the word "no"; but, to my chagrin, when I asked him to utter it, there was nothing but an articulatory effort. However, on asking him the preposterous question, "Are you ninety years old?" the word "No" came out at once. Again I asked him to say "no"; his efforts were fruitless, but we readily got the word out of him again by asking him another question, which obviously required a reply of dissent. His difficulty was not from nervousness; his wife had found out, before I did, that he could not say "no" when he tried.[1]

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[1] If a speechless man retains the two words, it is reasonable to suppose that the emotions of fear and anger would, to speak figuratively, appropriate the negative one, and that emotions of joy and sympathy would appropriate the affirmative.

I find that I have led Kussmaul to misunderstand me on this matter. This distinguished physician writes "Jackson and Sir Thos. Watson have even found that aphasic persons, unable to reply 'no' to a question, have nevertheless been led to do so by suggestions designed to make them angry, e.g. 'were they a hundred years old, or a thousand?' or such like." The questions were not designed to make the patients angry, and did not make them angry. The patients I speak of could reply by "no," at any time. A very preposterous question was asked in order that there might be no possible doubt that a negative was required; and I submit that the rejoiner "no" to such a question by the patient "Dow" was a reply, that it was "no" as a proposition; that it was speech, although inferior speech (not incomplete speech); and that it was not "no" as a mere utterance, like an oath coming out in anger. This patient, as I have stated, did use the word emotionally when vexed; but not so in rejoinder to the preposterous questions I asked him.

(*c*) In many cases of loss of speech, the patient, besides having the emotional service and also the power of reply with the words, can say "yes" or "no" when told (he has the full use of these words). It may be said that this third degree of utterance of the word is not speech; that it is uttering the word as an articulatory gymnastic "for the sake of uttering it," not *using* it as a proposition. There are weighty reasons, however, for drawing attention to the three degrees of utterance of this word. The inability to say "no" when told, with ability to utter it in reply and also emotionally, is one of the most important facts in the matter of affections of speech. I shall speak on this matter after considering analogous peculiarities.

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[1] The following is from the "Hospital Reports" of the *British Medical Journal*, December 2, 1871 [20], and refers to a case of loss of speech; ". . . She was told to say 'no,' and could not. Directly afterwards, Dr. Hughlings-Jackson, observing she had a book on her lap, asked if the patient could read. Hearing this, the patient herself looked up and said 'No, no, no.' She was again told to say 'no'; she could not. The nurse, having observed this peculiarity in another patient, said, 'Are you a hundred years old?' the response was 'No,' with a smile. Once more the patient was asked to say 'no,' but again she failed. . ." It must be added that in her ordinary "conversation" she sometimes said "no," when she meant "yes" In some cases there is no difficulty in saying "no." Thus, there is in the London Hospital an old woman who can utter only the words "yes" and "no," and "titty," "titty," "titty"; and another old woman who has both these words and the utterance "I'm very well," or "Very well." Each of these patients can say "no," when asked. Hence it is admitted that some aphasics have the full use of these words.

To resume. In some cases called *loss* of speech there is not absolute loss. The utterances (1), (2) and (3) are not exceptions; the utterances "yes" and "no" in reply, are exceptions, these words being *used* as propositions.

These exceptions are very significant. The man has lost all speech, except the two most general, most automatic, of all his propositions. They are indeed very significant exceptions to the empirical division into loss of intellectual and conservation of emotional language; for, even regarded superficially, they stand on the border ground. These words are used by healthy people, now one way, now the other; they are sometimes parts of emotional manifestations, and may then be combined with an ordinary interjection as in "oh! yes," or be duplicated as "no, no"; here the second "no" at any rate is interjectional. They are at other times used with full and definite propositional intent to signify "this is so," or "is not so." The word "yes" may be used at the same time, both for sympathy and agreement, it being occasionally hard to say whether the intellectual or the emotional side is more visible. Similarly some movements are at once pantomimic and gesticulatory.

We hear these words used almost purely emotionally, very often. A woman suffering from pleurisy and in great distress, replied "No" to a question, used the word propositionally, and then went on uttering the word as a vehicle of tone, "no, no, no," in just the same way as she had been uttering "oh!" before the question; that is, it served her emotionally. A healthy man told suddenly a piece of startling news, cries "No!" using "no" not actually to deny the truth of the statement; indeed he does not use it as a proposition; it is an ejaculation of surprise, equivalent to the exclamation "Nonsense!" or "You don't say so!" These so used are interjections, not speech, and take low rank in language, little above that of bodily starts, parts of common emotional language. At the best they are propositions, entirely subordinated to the service of an emotion.

Then propositionally "yes" and "no" give assent or dissent to anything whatever; they are the blank forms of, or stand for, all negative and positive propositions—are, as it were, propositions almost reduced to positive and negative copulas. From their almost universal applicability they are very frequently used; they are the most general, most automatic, and most organized of all propositions. They are then exceptions proving the rule; the patient has lost all speech except these two propositions which are at the "bottom" of intellectual language and at the "top" of emotional language. In other words, the retention of these two words is not exceptional to the principle of dissolution. The reader may, however, urge that the other recurring utterances are exceptional. I hope to show, later on, that they are not. At present I only say that I believe them to represent what was, or to represent part of what was, the last proposition the patient uttered or was about to utter when taken ill.

(2) OCCASIONAL UTTERANCES.

These utterances are rare, except that some patients swear very frequently. We shall make three degrees of these ejaculations:—

(1) Utterances which are not speech.

(2) Utterances which are inferior speech.

(3) Utterances which are real speech.

(1) Under excitement, the speechless man may utter "Oh!" or "Ah!" More than this, he may swear or utter certain nasty words used by vulgar people when excited. (We use the term "swearing" in the wide sense of what is popularly called bad language; of course religious commination is not considered.) The occasional utterance may be an innocent ejaculation, as "Oh dear!" or "Bless my life!" None of these utterances are speech; they have no intellectual meaning. Moreover, the patient cannot repeat them when he tries, he *utters* but does not *say*. This will remind the reader of what was said of some patients who can reply "no," but cannot say that word when they are told to try. The patient "Dow" uttered the word "damn" one night when vexed on his daughter coming in very late. Her mother told me of their surprise, and that her daughter said she would stay out late every night to get him to speak. But he could not repeat the expression. I never heard of any utterance in his case but of "damn" on that occasion, and his recurring "no" and "eh" (yes).

As said above, it is not a question of oaths only, but of ejaculations in general (interjections simple or compound).[1] They are all parts of emotional language; their utterance by healthy people is on the physical side, a process during which the equilibrium of a *greatly* disturbed nervous system is restored, as are also ordinary emotional manifestations. (All actions are in one sense results of restorations of nervous equilibrium by expenditure of energy.) In some people oaths and vulgar interjections have become very deeply automatic; some people swear largely along with their ordinary unexcited speech, perhaps to give emphasis to commonplaces. In these people the oaths are almost as automatic (their nervous arrangements being strongly organized) as smiles and frowns; they are, so to speak, "detonating commas." No wonder that they are occasional utterances when these patients are speechless. Few women swear, but their ejaculations of surprise or vexation (feminine oaths), as "Oh dear!" "Dear me!" "How very tiresome!" belong to the same category. The aphasic woman, whose recurring utterance was "Me, me," &c., once ejaculated, "God bless my life!"

(2) There are occasional utterances which are real speech, but inferior speech. This remark may be indefinite, but illustrations will show what is meant.

I saw, in consultation, a patient who had the recurring utterances "no" and "what." This patient was heard by his doctor to say "Wo, wo!" when standing by a horse. This patient once uttered "That's a lie," which is an expression often used by vulgar people as a verbal missile, that is emotionally rather than propositionally; it therefore comes under No. 1. The utterance "Wo, wo!" is the one I wish to draw attention to now. "Wo, wo!" is a proposition to those who use it, if not to animals; it means "stand still."

A woman who could only utter the phrase "Yes, but you know," once said "Take care!" when a child was in danger of falling. A patient of Trousseau's said "*Merci*" when a lady picked up his handkerchief. A patient of mine, would, besides swearing when vexed (No. 1), say "Good-bye," when a friend was leaving him.

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[1] I take the following from an unsigned review in the *Journal of Mental Science* for April, 1878, p. 125: "The value of swearing as a safety valve to the feelings, and substitute for aggressive muscular action, in accordance with the well-known law of the transmutation of forces, is not sufficiently dwelt on. Thus the reflex effect of treading on a man's corn may either be an oath or a blow, seldom both together. The Scotch minister's man had mastered this bit of brain-physiology when he whispered to his master, who was in great distress at things going wrong, 'Wad na an aith relieve ye?' It has been said that he who was the first to abuse his fellow-man instead of knocking out his brains without a word, laid thereby the basis of civilization."

The man who said "Wo, wo!" could not repeat it; and the lady could not repeat "Take care!" The friends of Trousseau's patient thought he was beginning to speak; but he could not repeat the word. My patient could never say "Good-bye," except under the appropriate circumstances; his daughter had found this out herself.

The following is from a communication made to the *Lancet*, [*vide* p. 105] on this patient's case. Several different phenomena are mentioned. They are all of the same order, in so far that they show conservation of automatic with loss of voluntary action.

"I have seen a patient who usually sat up in his room, whose face looked intelligent, who was cheerful and merry, and who seemed to understand all that I said to him, but who could not put out his tongue when he tried. His daughter remarked that he could put the tongue out, as she expressed it, 'by accident,' and added, as an illustration of her meaning, that when anyone was leaving him he could say 'good-bye,' but that he could neither put out his tongue, nor say 'good-bye' when he tried. He could say 'yes,' and 'no' at any time; and, using the lady's expression, could say 'good-bye' 'well,' 'never,' *by accident*. She further remarked that the patient would sometimes swear. He uttered the short explosive word which is so much in favour with English swearers, but he could not, she said, repeat the word when he tried. She asked him to utter the explosive sound when I was there, saying it herself for him to imitate. He laughed and shook his head."

Admitting the utterances (No. 2) to be exceptions, we have to note that, as exceptions, they are significant. They are true speech, but they are inferior speech. Superiority in speech does not mean number of words, nor even solely precision of application, but precision of application to new relations of things, that is, in effect superior speech is accurate speech on complex[1] matters. We do not find that the loquacious person speaks precisely, except on the most familiar things; on novel things he fails greatly. The "faculty" of speech is not, as popularly supposed, highly developed in him. The utterances are well organized; they were prompted—to speak popularly, helped out—by their special circumstances. They are only in degree less significant than the occasional utterances (1), or than the recurring utterances (4). Besides this, the inability to repeat them is to be carefully borne in mind.

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[1] Although of necessity we take type cases, we not only consider what we call exceptions to the type, but in actual practice we consider individual peculiarities. What is well organized in one person is not so in another; when we say that the more automatic, more organized, &c., remains, we mean what is more organized in this or that patient. The qualifications to be understood in using such expressions as "*the* concept," "*the* English language," "*the* environment," need not be pointed out. In such expressions as "from *the* special to *the* general," "from *the* complex to *the* simple," the obvious qualifications must be kept vividly in mind. When we speak of complexity of any actions, we do not mean any sort of abstract complexity. A man in delirium goes through very complex manipulations of his trade, but they are not complex to him.

(3) I have records of still higher degrees of utterance by one speechless patient. A man, for several months under my care in the London Hospital, was absolutely speechless. He never *uttered*, much less spoke, anything but "pooh," "pooh," so far as I or the students or the nurses knew. But I was told by his friends of three utterances. Once, when he had had enough bread-and-butter, he said "No more." This, however, is only a degree of speech on a level with those in the just-given illustrations (2). But I was told that one day the patient said, with difficulty, "How is Alice (his daughter) getting on?" A third utterance was, I think, as high, if not still higher, in speech. His son wanted to know where his father's tools were. In reply to his son's questions, the patient said "Master's." Although here is but one word where in health there would have been a sentence, there is a proposition; it told his son where the tools were as fully as the most elaborately worded and grammatically complete sentence would have done. It was far higher than the most elaborate oaths and higher even than such utterances as "no more," "good-bye," "very well," &c. Once more I would urge that speciality in speech ("high speech") is not simply an affair of number of words, nor simply of complexity of their arrangement. We have to consider precise adaptation to special and new circumstances; "master's" did not come out upon a common and simple occasion like "good-bye," it was definitely uttered to signify a very special relation, moreover a new relation. Granting for the sake of argument—which, however, I do not know—that the man had in health replied scores of times to the same question by that word, or by a fuller proposition containing it, it was specially used for a new occasion under, that is, very new circumstances. The father had left his work, would never return to it, was away from home, his son was on a visit and the question was directly put to the patient. Anyone who saw the abject poverty in which the poor man's family lived would admit that these tools were of immense value to them. Hence we have to consider as regards this and the other occasional utterances the strength of the accompanying emotional state. We shall consider the influence of strong emotions which imply great nervous tension on the production of these utterances later on.

I used to receive reports of these utterances and sayings by speechless patients with great incredulity, and so I find others have done. One of my most intelligent pupils, to whom I was speaking of such utterances, told me that the wife of a speechless patient, with much indignation at his (the student's) incredulity, affirmed that her husband (otherwise speechless since his attack) uttered, before he died, "God bless you, my dear!" This utterance, if largely emotional, was a painfully appropriate one from a dying man to his wife. He may have uttered it interjectionally scores of times when well, and have *said* it when dying; really *meaning* that God should bless his wife. Under some circumstances truths that have died down into truisms become alive again.

The Communist orator who began his oration by "Thank God, I am an Atheist!" used "thank God!" as a mere expletive. Even when this phrase is uttered devoutly, it is often more emotional than propositional; but in some states of mind it doubtless is *said* with full propositional intent. Much poetry, in prose and verse, nowadays seems to be an attempt to show the truths of what have become uninfluential truisms.

These utterances naturally surprise the friends of speechless patients. A patient, fatally ill, unable to tell what she wanted (this patient had not entirely lost speech), surprised her sister by exclaiming, "Surely you must know what I mean!" after that she said nothing intelligible. A patient under my observation in the London Hospital could utter many words, but his oaths and other ejaculations were alone properly uttered; a patient in the next bed felt insulted on being asked to note what the patient uttered. Naturally he would feel that a man who, when asked to write, ejaculated, "What's all this bloody nonsense about?" could talk if he liked.[1]

Gairdner had an aphasic under his care in hospital, and wishing to learn something as to the patient's general condition, asked another man in the ward what *he* thought of him. "I think a guid whuppin, wad be the cure of him." On Gairdner remarking that the patient could not speak, the man replied, "Na, but he swears whiles"; evidently believing that the poor fellow was shamming. The aphasic died, and cancer of the brain was found at the necropsy.

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[1] Dow's wife told me that the neighbours were very unkind; they said it was all nonsense about his being unable to talk, for why did he not write? They could not be expected to know that if speech goes, writing goes—expression in writing is meant; Dow copied a good deal, and could sign his name without copy. Had they heard Dow utter "damn" because his daughter came in late, they could have felt the correctness of their opinion of his case to be demonstrated. In general the laity cannot be expected to know that swearing, &c., may persist when speech proper is impossible, and certainly not that a higher kind of utterance may persist when the patient is fatally ill. No doubt many apoplectic persons found in the streets are locked up for drunkenness because the policeman does not know that swearing is a very automatic process, which can persist under conditions produced by fatal brain lesions as well as by drink.

In some cases of speechlessness, an elaborate utterance comes out of which we cannot guess the meaning. The following case is an illustration of this, and also of other utterances. A patient under the care of Dr. Martin, in St. Bartholomew's Hospital, could only utter the word "yes." The Sister of the ward (a very intelligent lady) remarked that he uttered this word when he meant "no"; moreover, she said he often nodded when he meant "no." One of the nurses told her that the patient once, in words, asked for beer; but the Sister remarked, "I don't believe this, as I was constantly with him, and never heard him say anything." I think it very likely he did, under strong excitement (active desire), get out a proposition to that effect. It may be said that there could be no excitement about so small a matter. But it is no small matter to many hospital patients. Some will leave the hospital if they do not get beer. But the utterance I wish to draw attention to is the following. His wife said that all she ever heard him utter beyond "yes," was "Five nights, six nights, seven nights, and then five nights out of seven." What this meant she could not guess.

To resume once more. There are three exceptions to the statement that our "speechless" man is absolutely speechless. He may have permanently the utterance of the words "yes" and "no," and the full use of them: their use as speech. On the other hand, we have noted that a patient may have only the emotional or interjectional use of them, and that when he has, more than this, the ability to reply with them, he may be unable to say them when told. And where there is the full use of them, we have to bear in mind that they are the most general of all propositions. Then he has *occasionally* some inferior speech, and as I believe this to be effected by the right half of his brain, I admit that these occasional utterances show, as do "yes" and "no," some power of speech during activity of that side. Anyhow, they show that the patient retains organizations for some words somewhere in his nervous system.

There is no demonstration by these cases that the patient retains organizations for any other words than those he actually utters. But is it a likely thing that Trousseau's patient, who said "merci," when a lady picked up his handkerchief, had just that word or a few such words left? A fire occurred in the street opposite one of my wards in the

London Hospital: a speechless patient of mine cried out "Fire!" Is it not a grotesque supposition that this woman retained only the word "fire"? Moreover, those who say a patient tried to repeat any of his occasional utterances are tacitly admitting that the words of those utterances are revived in him; otherwise the word tried has no meaning. There is demonstration by other means that the speechless patient retains a full service of words; he understands what we say to him. At any rate, the utterances spoken about show that there is retention of some words, if only a fragment or so in each case. Some of them also show that there is not only retention of some words, but of some speech, by the right side of the brain. The division we made was not that the left half of the brain serves in speech, and the right in receiving speech and in other ways, but that "nervous arrangements for words used in speech *lie chiefly* in the left half of the brain," and "that the nervous arrangements for words used in understanding speech (and in other ways) lie in the right also. It is believed that the process of verbalizing and every other process is dual, but that the more automatic a process is, or becomes by repetition, the more equally and fully is it represented double in each half of the brain. But the utterances show too, for the most part, that the speech possible by the right side of the brain is inferior speech. In nearly all cases it was well organized, automatic or "old," and nearly every utterance required a special occasion; was, to speak popularly, surprised out of the patient by a sudden accustomed stimulus. And it is to be borne in mind that the patient cannot repeat, say voluntarily, what he thus utters. So far these exceptions are exceptions proving the rule.

It has been admitted, however, that occasionally there is an utterance of high speech value. This exception will be considered after a while.

ON AFFECTIONS OF SPEECH FROM DISEASE OF THE BRAIN.

[BRAIN, 1880, vol. ii, p. 328.]

IT will doubtless have struck all physicians as strange that speechless patients should have such recurring utterances as jargon No. 1 (p. 132), or single words, No. 2 (p. 132), or, what is most striking, such as "I'm very well," "Come on to me," No. 3 (p. 134).

It may perhaps be well to give some examples of these several kinds of recurring utterances from Trousseau's admirable lecture on aphasia. (I take them from Bazire's translation for the New Sydenham Society, adopting his rendering into English of the utterances of real words from the French; some of the utterances were jargon.) One patient could utter nothing whatever else than "Oh! how annoying!" Trousseau says of this patient: "Although she looked intelligent and behaved like a sensible person, I could never obtain another word from her." Another patient uttered, "Vousi, vousi." Another at first could only utter "Monomentif"; later on a few monosyllables ended by "tif," or the first syllable of a word and "tif" instead of the second, as "montif" for monsieur. Another uttered "Pig, animal, stupid fool," and that alone. Another "Nasi bousi, nasi bousi." Another, "My faith!" Another patient, "Cousisi, cousisi." (This patient, by the way, had the occasional utterance when very excited, "Sacon, sacon," which Trousseau supposed to be abbreviation of "Sacré nom de Dieu.") Another, only "Oh! mad." Another, "Sacré nom de Dieu."

It will have occurred to the reader that these utterances are not in the same category as "yes" and "no" in regard to the hypothesis stated (p. 140). We cannot say that the cases in which they remain show a reduction to the most general of all propositions. Some of the utterances of Group No. 3 are propositional in form, but yet not of use as propositions to the speechless man; they are to him not propositions at all, figuratively speaking, they are propositions intellectually dead; they serve only emotionally, and in that way subsidiarily as a sort of articulatory material on which the voice may vary. "Yes" and "no," Group No. 4 (p. 135), also are occasionally from the mouth of the speechless man, intellectually dead (see No. 4 (*a*), p. 135), and serve only emotionally; nevertheless these utterances so serving are in a different category from those of Group 3. We have even on this basis to ask, "How is it that some speechless patients retain an utterance of no propositional value to them, but one which to healthy people is a proposition of a *special* character, applying to a particular combination of circumstances, whilst other aphasics retain an utterance of no propositional value to them, but one which to healthy people is of propositional value of the most *general* character, applying to any combination of circumstances?" Of course, the utterance may be an elaborate (compound) interjection, as "Oh, my God!" but we prefer considering those utterances which in health are propositional.

The problem may be presented in one case. A patient has "yes" and "no," and has also some elaborate recurring utterance in propositional form. Supposing his "yes" and "no" to be utterable only emotionally, we may say that he has two intellectually dead propositions one of a highly special kind, another of a very general kind.

We wish in this instalment to show that the retention of recurring utterances other than "yes" and "no," although apparently, is not really exceptional to the principle of dissolution. In all cases of dissolution there is supposed to be a reduction to the more automatic, more organized, to the earlier acquired of the processes represented in the centres affected. It is, I think, plain that this course is in some cases apparently irregular. There are conditions interfering with what we may designate the simple course of dissolution.[1] Let me illustrate this by a case from another class of nervous affections. Just after a slight epileptic seizure there is dissolution, which in this case is temporary loss of function of more or fewer of the highest of all nervous arrangements; the patient acts grotesquely; but his actions (occurring during energizing of nervous arrangements next lower than those which have undergone dissolution) are not always rangeable as being more organized, &c., than his usual doings, sometimes they are plainly largely determined by particular events happening just before the seizure—that is to say, there are conditions interfering.

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[1] The reader is asked again to observe that the term "dissolution" is used, as it is by Herbert Spencer, for a process which is the reverse of evolution. I am sometimes told that it is not a fit term to apply to the results of disease on the nervous system, granting that those results are the opposite of evolution of the nervous system. I do not believe they can be shown to be the exact corresponding opposite. Even supposing that the term does not properly apply, I submit that it is inexpedient to coin another word for a process the reverse of nervous evolution, when dissolution is already in use for the reverse of evolution in general. Affections of speech are examples of dissolution occurring in a subordinate cerebral centre. Insanity, as for example the case of post-epileptic insanity mentioned in the text, is dissolution, beginning in the highest cerebral centres, in those centres which are supposed to represent all that the subordinate centres have already represented, and thus, indirectly, to represent the whole organism. The highest centres are the substrata of our highest, latest, ever-changing mental states, the abstract name for which is consciousness, there being really a series of consciousness.

The matter to be discussed is not of scientific importance as bearing on the principle of dissolution. The discussion will help us, I think, to clearer notions on the nature of defects of speech. Were recurring utterances alone in question it would not be worth a physician's while to consider them at much length. We shall in the next instalment consider other kinds of nervous disease in which we find phenomena analogous to the recurring utterances of speechless patients.

The following hypothesis is advanced, and for the present is applied to but one set of recurring utterances, the group No. 3 (p. 134), and only to those of them which have in healthy people propositional value. *Such recurring utterances as "Come on to me" were being said or rather were about to be said when the patient was taken ill.* Here we have, it is suggested, a condition interfering with the simple course of dissolution.

In order that we may show this hypothesis to be reasonable, the reader must bear vividly in mind that "taken ill" means here the occurrence of damage in a region of but one half of the brain, the left usually, sufficiently extensive to cause loss of speech. He must keep in mind that the nervous arrangements in this cerebral region are *destroyed;* it will not do to think of the disease vaguely as something "disordering certain functions of the brain." And on the psychical side he must look on the condition as one of *loss of the words* used in speech. The expression, 'loss of memory for words,' is too indefinite, or is only verbally definite. It is equally important for him to bear vividly in mind that the other half of the brain, the right usually, is not at all injured. He is asked never to lose sight of the fact that, although the patient is rendered speechless by the disease, he is not thereby rendered wordless. We must, indeed, bear most vividly in mind that *the patient has words remaining;* it will not do to think of this positive condition under the vague expression that "he retains a memory of words." If we do use such redundant expressions, we must be thorough in our application of them and say two things: (1) that the speechless patient *has lost the memory of the words serving in speech,* and (2) that *he has* *not lost the memory of words serving in other ways.* In healthy people every word is in duplicate. The "experiment" which disease brutally makes on man seems to me to demonstrate this; it takes one set of words away and leaves the other set. The speechless patient has *lost* that set of words which serves in speech; he *retains* another set of words serving in other ways;[1] he *retains* all the words of the (his) English language in so far as "receiving speech" of others goes; and for other purposes he has *lost* their duplicates by which he should speak. It is suggested that whilst the speechlessness answers to the damage in one half of the brain, the retention of other kinds of service of words answers to the fact that the other half is not damaged. Attention is also asked to the hypothesis already stated, and hereafter to be expounded at length, that every proposition in health occurring during activity of the left side of the brain is preceded by a revival of the words of it during activity of the right half.[2] This is stating the hypothesis roughly and inadequately; all that need be insisted on is that the unit of action of the nervous system is double the unit of its composition, and that, correspondingly, all mental action is dual; subjective followed by objective. But, as roughly put, the hypothesis will serve our present purpose, since the speechless patient has no left half of his brain so far as its use in words goes.

Here brief remarks may be made on dual action in other mental processes in order to make clear what is the nature of the dual service of words. If I do not succeed in showing that there is a duality of mental operations and what is the nature of that duality, I shall fail to make clear my meaning as to the nature of recurring utterances and to interpret many other difficult parts of our subject. We take for illustrations ideation and perception; that is to say, "propositions of images," which are symbolized by propositions strictly so-called. This is truly a part of our subject. It is, indeed, an entirely arbitrary proceeding to separate the images symbolized from the symbols of images; in what we shall call the "conflict" both are concerned. Nevertheless the artificial separation of them is convenient in brief exposition. It will suffice for illustration to take count of but one image, although two images must be concerned in every ideational or perceptive process and also in the stage preceding ideation or perception (ideation may not be followed by perception, but ideation must always precede perception).

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[1] The use of such highly technical expressions as "memory," in "explanation" of complex symptomatic conditions, seemingly definite and authoritative, is largely to blame for our remaining with our ideas on nervous diseases out of focus. It is not always vividly realized that retention of memory of words can mean anything more than a retention of words, the word "memory" in that context being really surplusage. There is almost the idea that the speaker *gives* words to the persons spoken to; all the speaker does is to rouse words already organized in the person spoken to. Anyone will readily admit that the pain of a pin-prick is in himself, not in the pin; and every educated man admits that redness is in himself when he uses the ordinary language, "this brick is red." Why, then, should we speak of "retention of memory of words" by the aphasic when he understands what we say to him without, at any rate, using that expression as simply convertible with the expression that he retains words (physically organized nervous arrangements for words)? Whose words are those which occur in the so-called hearing "voices" of the insane?

[2] There are, then, it is supposed, two services of words by the right half of the brain: the "reception" of words of others, and the reproduction of words which precede our own speech. These are the two ways other than speech in which words are supposed to serve. Doubtless at bottom these two are alike.

It seems clear enough that the process which ends in ideation or perception is dual. *Before* we can see or think of a brick, before either ideation or perception can occur, that brick, by actual presentation or by indirect presentation through association, must have roused an image in us. When we say we see a brick, all we mean is that we project into the environment, ideal or actual, the image which that brick has roused in us. This image arisen in us is the survival of the fittest image at the termination of a struggle which the presented brick has roused in us, is the end of the subjective stage; the further stage, or the second stage, is objective—it is referring the image already roused in us to the environment, actual or ideal. The brick is for us nothing more than what it has itself roused in us, so that, instead of using such figures as that "the mind impresses its own laws on sensations we experience during contact with external objects," or "that the mind originates something additional to them," or "works them up into different shapes," or that it "impresses its own form on them," we should, I think, use the opposite figure that an external object acts on us and develops in us such as we are, *what it can*, we being at first passive and it active. I think it is plainly so in dreams excited by peripheral local excitations. And in reverie, cracks and marks in the burning coals make us see faces in the fire, rouse faces in us.

Returning to the dual service of words. If we coin the term "*verbalizing*" to include the whole process of which speech is only the end or second half, we may say that there are in it two propositions: the subject-proposition followed by the object-proposition, the latter being called speech. Spencer writes ("Psychology," vol. i, p. 162): "A psychological proposition is necessarily compounded of two propositions, of which one concerns the subject and the other concerns the object, and cannot be expressed without the four terms which these two propositions imply." It is supposed that the subject-proposition is the "survival of the fittest" words in fittest relation during activity beginning in the right half of the brain, and that this survival is at once the end of the subjective and the beginning of the objective stage of verbalizing. The subject-proposition symbolizes an internal relation of two images, internal in the sense that each of them is related to all other images already organized in us, and thus it symbolizes states of *us*. The object-proposition symbolizes relation of these two images as for things in the environment, each of which images is related to all other images then organizing from the environment; thus it stands for states of the environment; the two propositions together symbolize an internal relation of images in relation to an external relation of images. Thus the separation we have made into speech use of words and not speech use of words may appear arbitrary; it is, however, convenient.

In one of the cases to be presently mentioned in the text—the patient who said "Come on to me"—there was *left* hemiplegia, and thus the inference is irresistible that his speechlessness was caused by damage in the right half of his brain. But as he was a left-handed man, his case is an exception proving the rule. It is admitted that there are cases of left hemiplegia with aphasia in persons who are not left-handed. Besides granting fully the significance of the fact that in the vastly greater number of cases loss of speech is caused by disease in the left half of the brain, the thing of infinitely greater significance is that damage in but *one* half can produce speechlessness. It is equally significant that damage in neither half produces wordlessness.

In a few cases I can state the circumstances of the onset of the illness, which seem to me to countenance the hypothesis stated. The man whose recurring utterance was "Come on to me," and sometimes only "Come on," was a railway signalman, and was taken ill on the rails in front of his box. A woman in Guy's Hospital could only utter "Gee gee"; she was taken ill whilst riding on a donkey. Dr. Russell, of Birmingham, has published the case of a clerk, who lost speech and became paralysed on one side, after hard work in *making a catalogue;* this poor fellow could only say "List complete." Sir James Paget a few years ago had under his care in St. Bartholomew's Hospital a man whose left cerebral hemisphere was injured in a brawl. This man could only say, "I want protection."

The hypothesis further unfolded, but applied only to the above-mentioned cases for the present, is that the words of the recurring utterance had been revived during activity of the right half of the brain, when the destruction of that part of the left half occurred which caused loss of speech; that they constituted the last proposition, or rather that stage of verbal revival (what we have called the subject-proposition), prior to the last proposition (the object-proposition). Even after they were meaningless or, figuratively speaking, dead propositions, remaining utterances of which we can only say they were on one occasion speech; on that occasion fitted to indicate then occurring relations of things. On the rails, or certainly at some juncture, "Come on to me" *meant* "Come on to me," ever after the illness it meant nothing.

The expression "on one occasion" is not to be taken literally as meaning that; for example, the man who uttered "Come on to me" never said that before he was taken ill on the rails. The presumption is that he had said it on many occasions, when trains were approaching his post. What I mean rather is, speaking now of the healthy, that there is no such proposition in a man's mind, excepting when he is saying it. On the physical side we would suggest that the nervous arrangements concerned during the utterance of any such proposition are not excited, nor ready to be excited, in the particular order answering to such proposition except at the time of uttering it. Such propositions are new speech. The "same" proposition is new or "latest" speech each time it is uttered, if the words or syllables of it are not, so to speak, "kept ready made up" in that particular combination; at any rate it is "latest speech" when specially applied at a particular time to indicate then occurring relations of things which *are not fully organized*, it is then, otherwise stated, a voluntary use of words.[1]

From the most automatic or "oldest" or "earliest" speech, applying to well-organized external relations of things up to newest speech, applying to now organizing external relations, or otherwise put propositions now in the making, there are, it is supposed, on the physical side, only degrees of independency of organization in the sense that the nervous centres concerned have lines of different degrees of resistance to currents of different force, entering the centres at different points. Other things equal, the more automatically serving (the more organized) nervous arrangements are lines of least resistance. There is, however, in the case of very frequently used and very automatic utterances, such as "Very well," "I don't know," a near approach to independent organization, and in the case of the most common interjections a very near approach to it. Such utterances constitute what I call old speech, or, in the case of interjections, they are verbal processes lower and earlier than true speech—utterances not "now making," but nearly, if indeed not quite, "ready made up." And at the other extreme, in the highest, newest, or latest speech, there remains for a short time after the utterance a slight degree of independent organization of the nervous arrangements concerned; did there not, we should not know what we had just said, and could not go on talking consecutively on any subject. If this reasoning be correct, such a recurring utterance as "Come on to me" represents not only the last proposition *en permanence*, but it is to be looked on as on the physical side, a keeping up of activity (of a greater readiness to discharge), of certain nervous arrangements, which normally exist only temporarily and only on special occasions, in particular combination, and which in health go quickly into subordinate function, and soon out of function.[1]

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[1] The very same "utterance" which has *become* automatic by being often used for symbolizing frequently presented, and therefore well-organized, external relations of things would be once more a voluntary utterance, if it were used on a new occasion, that is for symbolizing a relation of things not already organized, but only now organizing. The "highest," "latest," or "newest" speech (the now organizing or voluntary use of words), implies clear preconception; clear preconception is a necessary element for voluntary, as distinguished from automatic, operations; in voluntary speech the prior reproduction of words constitutes the preconception. If a man utters, as applying to a new set of circumstances, the most automatic utterance he has, or if he utters anything "for the sake of uttering it," as when asked to do so, there is then a voluntary utterance, for then the operation occurs after clear preconception. Disease shows this not to be a fanciful dictum. As we have seen, and we shall later on give further examples, a speechless patient may be unable to *say* (to repeat) what he has just uttered. We do not affirm that the word "say" in this context means a language process.

Such a recurring utterance as "Come on to me" was about to be new speech; the words remained *en permanence* as a dead proposition; a proposition stillborn. We can now describe the case of a man who has two sets of recurring utterance as that of a patient retaining a new combination of words, and also an old combination, or rather in the latter two monosyllables, each of which is equivalent to a combination of words.

The reason for thinking that the recurring utterances under remark were and continued to be utterances occurring during activity of the *right* half of the brain, is that the left half of the brain is so extensively damaged that the patient cannot speak; he has no left half for their utterance. To say that the disease "caused" these utterances—a positive condition—is absurd, for the disease is destruction of nervous arrangements, and that could not cause a man to do something; it has enough to answer for in leaving him unable to speak. The utterances are effected during activity of nervous arrangements which have escaped injury. This remark may seem a truism here, but in more complicated cases it is very common to hear of positive symptoms being ascribed to negative lesions—to loss of function of nervous elements. It is common at any rate for disease to be thought of vaguely as something "*disordering* the functions of the brain." In the cases we are now dealing with, and in cases of defect of speech, and also in cases of insanity, the function of some nervous arrangements *is lost or is defective.* But it is an error to ascribe such positive symptoms as the recurring utterances in speechless men, the erroneous words uttered by those who have defect of speech, and the hallucinations, &c., of insane persons, to negative lesions, to loss or to defect of function. These positive mental symptoms arise during activity of lower centres or lower nervous arrangements which have escaped injury, and are only to be thought of as symptoms in the sense of being the fittest psychical states arising during slightly hypernormal discharges of lower or more organized nervous arrangements which are *then* highest, the normally highest having lost functions.

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[1] Practically out of function, so far as our present subject is concerned, although, obviously, there would be no new acquirements possible, and no memory, if some nervous arrangements after their use did not remain lines of less resistance than before.

It might be suggested that not all, but only nearly all, the substrata of speech were destroyed, and that enough nervous arrangements remained in the left half for just one utterance. Since, as implied in a foregoing statement, I do not believe that words or syllables have nervous arrangements in the sense that there is one little nervous centre for each syllable, or rather single articulation,[1] and for no other, and since, too, I believe that each unit of every nervous centre is the whole of that nervous centre in (different) miniature, I should be as ready as most people to accept this explanation. But a certain quantity of nervous arrangements, implying a certain quantity of energy, is required for every operation. As the recurring utterance is, as a series of articulations, very elaborate, and as the syllables are clearly enunciated at any time, it is not credible that slight remains of nervous arrangements can be concerned during the utterance of them, especially when looking at the matter on another side we see that these remains do not serve the patient to utter any other words whatever, except perhaps "yes" and "no," which I, however, believe to occur also during activity of the right half of the brain. The man who uttered "Come on to me" uttered it at any time, with no effort, rapidly and in fact just as well as a healthy person could utter it; yet except for this and "yes" and "no," he could utter nothing else. It is submitted that it is not possible that such an utterance was effected during discharges of remains of a nervous centre so extensively diseased, that is to say, not during discharges of any part of a half of the brain which was damaged so much as to cause speechlessness. It is more likely to be uttered during activity of the undamaged half.

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[1] The reader will observe that nothing is said in the text which implies that a word *is* a nervous arrangement representing an articulatory movement. All that is suggested (see p. 108) is that the *anatomical substratum* of a word is a nervous arrangement representing one or more particular articulatory movements. The reader will observe, too, that we sometimes speak of words, and at other times of their physical bases, but we do not, it is hoped, confuse the two different things.

We repeat, the patient may have not only such a recurring utterance as "Come on to me," but he may have also the utterance "yes" or "no," or he may have both these words. We have endeavoured to explain the retention of "yes" and "no," by saying that the patient is reduced to the two most automatic, the two most organized, of all propositions. We have tried to show that in a sense the principle of dissolution is not contradicted by the retention of the elaborate recurring utterances under remark. There are, we have suggested, conditions interfering with what we may call the simple course of dissolution. The patient retains not only his most automatic propositions, but his last proposition, the then most voluntary proposition; the former sometimes, the latter always, ceasing to be propositions.

Let us now look more particularly at the anatomico-physiological side: that is, examine the utterance from the side of degree of organization of nervous arrangements. We must bear in mind that, to speak metaphorically, all disease cares about is degree of organization; it respects processes the more they are organized, caring nothing about important or unimportant. At present we can only speak in outline.

In estimating organization in its bearing on affections of speech, or on any other sort of defects from nervous lesions, we have to consider recency of organization, as well as degree of organization. It is well known that in failing memory recent events are soon forgotten, whilst old events are remembered; but this is not without qualification; the *most* recent events are remembered as well as the old; on the physical side, nervous arrangements just discharged remain for a short time in a state of slight independent organization, rivalling that of nervous arrangements discharged when the person was young and vigorous, or that of nervous arrangements often discharged.

That the nervous arrangements for the words of the recurring utterance (No. 3) are parts of the patient is a truism; that the words must at one time have been revived in propositional form, that certain nervous arrangements must at one time have been discharged in a particular order, is another. The hypothesis that the words were being revived at the time when the patient was taken ill seems to me warrantable. As they remain always easily utterable, and they alone utterable, since the illness, it is plain that certain nervous arrangements which were being discharged when the patient was taken ill, or at any rate which were on some occasion being discharged in a particular definite order, remain permanently in a state of dischargeability far above normal. For the patient not only does not speak since his illness, but whenever since his illness he tries to speak, the recurring utterance comes out or nothing comes out. Note three equally important things: (1) He has it; (2) he has no other utterance (except perhaps "yes" and "no"); (3) he cannot get rid of it. It is certain that the nervous arrangements for the recurring utterance have somehow arrived at what is in effect a high degree of independence of organization. We can say, if the hypothesis put forward be true, that the nervous arrangements for the proposition *just organizing* at the time of the illness, those for the words last spoken or about to be spoken, remain as well *as the most and always organized* nervous arrangements for the most automatic propositions, and have somehow achieved a degree of independent organization greater than that of any other word-processes except "yes" and "no"; probably by repeated utterance after their first utterance. On this matter we shall speak later, when considering the phenomenon called by Gairdner "barrel-organism." At present the explanation given may seem to be merely a verbal one. We may consider part of the question at once. Repeating that the patient has the utterance, and that he cannot get rid of it, we restate the question thus: How is it (1) that destruction of one centre (certain part of the left half of the brain) leaves in state of hypernormal dischargeability particular nervous arrangements discharging in another centre at the outset of the illness, right half of the brain, a lower centre? And (2) How is it that they do not go out of function, as they certainly would have done had not that destruction occurred? We say again that the destruction in the left half is not the cause of the recurring utterance; a negative state of nervous elements cannot possibly be the cause of positive nervous symptoms. Nor is destruction of some nervous arrangements the cause, or at any rate not the direct cause, of certain other nervous arrangements discharging and remaining in a state of hypernormal dischargeability. But plainly, if the man had not been "taken ill" he would not have had such a recurring utterance; he would have been able *not to utter it*, the nervous arrangements concerned would have gone out of function; but since his illness, he must utter it when he tries to speak, if he utters anything. I believe the solution of the seeming paradox to lie in this, that destruction of function of a higher centre is a removal of inhibition over a lower centre the lower centre[1] becomes more easily dischargeable, or popularly speaking, "more excitable," and especially those parts of that centre which are in activity when control is removed. So to speak, these parts become autonomous, acting for themselves, just as parts of the spinal cord below a diseased point become autonomous in some cases of paraplegia. Thus the disease *causes* loss of speech; it *permits* the increased dischargeability of the right half. It is just as cutting the pneumogastric does not cause, but permits, increased frequency of cardiac beats. But, the simple course of dissolution being interfered with in the cases of speechlessness we are writing about, the activity of the centre manifests itself in the way it was temporarily in when control was removed. The greater excitability of a centre uncontrolled by removal of its higher centre is supposed to be temporary, but the particular activity may be kept up by repeated use; the speechless man's recurring utterance is being uttered very often; it keeps up what was doubtless at first a temporary organization.

We must be very careful how we use the word "cause" with regard to disease and symptoms. We must never speak of destructive lesions causing positive symptoms. It is erroneous, I submit, to say that any sort of disease causes elaborate positive mental symptoms—illusions, hallucinations, &c.—it causes a negative mental condition, the elaborate positive mental symptoms are *permitted*. Here again we see the crudity of the expression, "disease disorders the function of the brain"; destructive lesions cause loss of function of some nervous arrangements, and thereby over-function of others is permitted. Another way of putting this is to say that the effects of dissolution are not always, never I suppose, simply those of removal of the more special, voluntary, and least and latest organized, but are very often also a permitted increased activity of the next most special, voluntary, and next least organized. Thus in every case of insanity there *are* two diametrically opposite symptomatic mental states together, and there must be correspondingly the opposite physical conditions together.

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[1] Of course, we do not mean geographically, but physiologically lower, more organic, &c., and, in some instances, the centre which acts before the one called higher.

In some cases there is the utterance of "yes" or "no," or of both, without any other recurring phrase. This would be explained on the supposition that the patient was not saying, nor about to say, anything when taken ill, and thus that there were no words being revived, no proposition organizing, during activity of the right side of the brain. Thus there was, so to speak, nothing to interfere with the simple course of dissolution, reduction to the most automatic of all propositions.

It is to be clearly understood that in not one of the cases do I know what the patients are saying, or about to say, at the time of their attack. I must also mention that there are observations on record which perhaps run counter to the hypothesis. Thus a man, aged 21, suddenly called out to his mother, "Oh! I feel something extraordinary inside me." These were the last words he spoke, but his recurring utterances were "no" and "Mamma." Very likely, however, calling out to his mother, he would also have cried "Mamma."[1]

I must also declare that of other cases than those expressly mentioned, I know nothing at all of particular circumstances during the onset of the ailment, which could be supposed to have given rise to speech like the recurring utterances the patients had. I have no facts to bring forward to show what were the particular circumstances during the onset of the illness of the patients whose recurring utterances were severally "man," "one," "awful." "Yes, but you know," "I'm very well," &c. At first glance it seems unlikely that a patient should say, "I'm very well," at the time she was taken ill; but as a matter of fact, people becoming ill do make such remarks. Who has not heard a person say, when a little ill, or when a severe illness was beginning, "I'm all right, let me alone," perhaps adding the contradictory statement, "I shall be better directly"?

We must bear in mind that when people are not talking they may be speaking, for there is not only external, there is also internal, speech.[2]

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[1] I take this case from a lecture by Trousseau, the utterances were in French, but I give them as translated by Bazire.

[2] The reader will remember that the distinction into internal and external speech (only a difference of *degree*) is not that made into the two *kinds* of service of words, the subjective and objective. There are degrees of each kind. The subjective, at any rate in that part of it which is "receiving speech" of others, is plainly in degrees, we hear and "receive" what people are actually saying, we also remember what they have said, which is having again, faintly, the words in ourselves which their speech had revived strongly. It must never be forgotten that when we hear and understand what anyone says to us, we can only do so because our own words are revived in us; if I have not the words "gold," "is," "yellow," organized in myself, it is of no use any one saying "gold is yellow" to me.

Whilst thinking on anything, at least beyond the simplest, we are using words, and when thinking on anything complex, are speaking internally. But I do not see how we are to verify the hypothesis that a patient's recurring utterance was a survival *en permanence* of the words he was internally using, unless perhaps the circumstances and the conditions of the patient are known very exactly.

We have to consider how the hypothesis applies to the recurring utterances, No. 1 and No. 2. How is it that there is (except perhaps "yes" and "no") jargon only, or but one word only? The difficulty is greatest with regard to the jargon (No. 1), and this I shall chiefly consider. I believe the jargon to be made up of fragments of the words or phrases the patient was about to utter when taken ill.

It is, I grant, a mere guess that a speechless woman, whose recurring utterance (I spell it from the sound) was "me" "me," "pittymy," "committymy," "lor," "deah," was saying, when she was taken ill, "Pity me," "Come, pity me," "lord," "dear"—that her recurring jargon-utterance represented what was part of the verbal revival prior to her last propositional or interjectional utterance, that it was a corruption of syllables of some of the words which were being revived during activity in the right half of her brain, when destruction in the left half produced loss of her speech. There is a greater difficulty in supposing that such a jargon-utterance as "yabby," resembling no English word, was made up of fragments of words which were being revived during activity of the right half of the brain, when the patient was taken ill. To say that "yabby" was a jumble of syllables of some words about to be uttered will appear to be carrying the hypothesis to its logical conclusion with more determination than caution.

Let us pause to remark on occasional temporary jargon, and on something approaching to it, in other aphasic cases. Some patients, who have only defect of speech, and whose erroneous utterances are nearly always real words, may occasionally utter "jargon," as "totano." I confess that I am unable to trace the steps of the formation of such jargon. Occasionally there is a very near approach to a transposition of syllables of a word. One of my patients said "gippin" for "pigeon."

Similarly the objective is in two degrees; there is both internal and external speech, corresponding to ideation and perception. Speech in *either* of these degrees is supposed to be preceded by an automatic and subconscious reproduction of words (by a subjective proposition), just as either "internal" perception (ideation) or "external" ideation (perception) is preceded by a subconscious and automatic reproduction of images. It is not meant that, either betwixt ideation and perception, or betwixt internal and external speech there are differences only in simple degree; the degrees are, I think, triply compound.

There is another kind of change: one of my patients said "lamb and crobster" for "crab and lobster." There is here a fusion of the first syllable of the second word with part of that of the first, or rather, perhaps, speaking of the physical side, a result of attempts at two articulations. Such blunders occur, I think, in persons whose speech is only very slightly defective: I mean in those who, for the most part, speak well. The following occurred in healthy people: "mukes from Boodie's," for "books from Mudie's"; "get a cash chequed," for "get a cheque cashed." A similar thing occurs in writing, as "Mear Dadam," for "Dear Madam"; "pred budding," for "bread pudding." (This is interesting, as showing that expression by writing is really speech.) I believe that these troubles of speech are owing to hurry on the right half of the brain, to hurried reproduction of the words of the subject-proposition. Believing that images and words are subjectively revived in an order the reverse of that in which images and words are finally arranged, I suppose, speaking roughly, that the words of the subject-proposition "come over" to the left side prematurely. These utterances are jargon, the sources of which we can trace, in so far, I mean, as fusion of an initial syllable of a second word with that of a first word goes. And I believe this to result from hurried action in patients who have defect of speech, partly for the reason that such and similar mistakes occur when a patient replies at once to questions abruptly put to him, and because when not hurried, when not "taken suddenly," he may speak for the most part well.

We can now return to consider more easily the permanent recurring jargon of patients who are speechless. We speak of what is supposed to have occurred in the right half of the brain, when the speechless patient was uttering, or about to utter, his last proposition. If the reader does not go with me in supposing that the right half of the brain is concerned, he must, at any rate, believe it is some part other than that on the left which the disease destroyed. It is suggested that when the recurring utterance is in clear propositional form, such as "Come on to me," the right half of the brain was acting normally or usually; that when it is a single word or jargon, it was acting abnormally, being "hurried." What is the cause of this hurry? I believe it to be strong emotion. At the time of the onset of the illness, we may suppose strong emotion would be induced by the external circumstances, or much more probably by the setting in of the illness, the patient being afraid of worse to come. Here, of course, a knowledge of the patient's temperament is very important. The patient who uttered "me," "me," "pittymy," "committymy," "lor," "deah," was, to my own observation, a most excitable woman. Her husband used to remark on this. He said she was fond of sensational tales; she would any time go out in the night to a fire. Strong, especially suddenly induced, emotion implies, on the physical side, great nervous excitation, that is to say, strong, wide, and sudden discharges; there will, under strong emotion, be more rapid and more numerous and therefore conflicting, discharges of nervous centres. In the cases under remark we consider the result of discharges on centres for words.

The result of numerous sudden, strong, and therefore conflicting discharges, would of course be the survival of the fittest, but the survival amidst too numerous and too strong discharges would not be an elaborate and perfect proposition. We suggest that it would be but one word or a jumble of syllables of some words. Of course "fittest" here does not mean "the best," nor the fittest *for* the *external* circumstances of the time; it is the survival of the fittest under the *internal* circumstances.[1] The fittest words of syllables are the victorious words or syllables, which have survived during a conflict of very strong and sudden discharges, beginning deeply in the nervous system, and ending, so far as we are now concerned, in that of many nervous arrangements for many words. Such survivals may be little fit or not fit at all to the then external circumstances, they express the emotions and ideas induced well or badly, as well as it can be done under the circumstances.

It is supposed that jargon is a survival of some of the syllables or articulations, more or less fused during the sudden termination or arrest of a strong conflict of discharges of numerous nervous arrangements for different words. It is a popular doctrine that strong emotion leads to incoherent utterances, which, I suppose, means the survival of words symbolizing more than one different set of ideas. Strong and suddenly induced emotion may lead to temporary speechlessness; we suppose that temporary speechlessness with great emotion betokens very numerous and strong nervous discharges, conflicting so as to balance or neutralize one another.[1] In some other cases of disease psychical states cease during sudden excessive discharges. An illustration is that during sudden and strong discharges, epileptic discharges, beginning in the very highest nervous arrangements (consciousness), the substrata of consciousness cease.

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[1] The words and images which survive during healthy discharges are survivals of the fittest. So it is in disease. The images that survive in delirium are the then fittest, considering that some of the highest nervous arrangements are *hors de combat*. The delirious patient is not the same person as that patient before his delirium set in; the delirious patient is the same man *minus* more or fewer of his highest nervous arrangements; his delirium is the fittest mentation then possible. In all cases it is supposed that what becomes conscious is what survives at the end of a conflict; the conflict is almost at zero in highly organized processes, such as those for recognizing very simple and very often seen things; in these cases consciousness is almost at zero too. In movement of any part of the body there is not only co-operation, but antagonism also of muscles or muscular strands (Duchenne uses the expression "co-operation of antagonism"). The antagonist pulls—those against the direction of the displacement to be effected—may be symbolized as minus quantities; the pulls co-operating in that direction as plus quantities. A movement is then not the arithmetical sum of pulls of all the muscles engaged, but the algebraical sum of the pulls Transferring these ideas to antagonistic and co-operating discharges of nervous centres, we may suppose that what we have called, both for health and disease, the survival of the fittest during a conflict of numerous nervous discharges, is the algebraical sum of what on the physical side are co-operating and neutralizing discharges during the conflict.

We now consider the recurring utterance of one word (No. 2, p. 132). Let us first note what occurs in health. Very often under strong and sudden emotion, a normal or usual degree of speech is not possible; some inferior speech is possible. When there is not incoherence, but one word may be uttered or rather exclaimed, as "Fire!" "Help!" the utterance is indeed often a very simple injection, as "Oh." One word, as "Fire!" might in normal circumstances be propositional only or so practically; it might *mean* "there is a Fire," but exclaimed under strong emotion it is also largely of interjectional value. Strong emotion leads to inferior speech, to more automatic, more organized utterance. Figuratively speaking, emotion uses propositions in a largely interjectional manner, that is to say, reduces them to or towards inferior speech. Emotion, as it were, still speaking figuratively, appropriates and subordinates an intellectual utterance. There is at any rate an interjection in the making, in the exclamation "Fire!" "Help!" &c., a degradation of speech (compare swearing). We see in the utterance itself, that with a fall in the intellectual element there is a rise in the emotional one. There may be an equal, no doubt a greater, liberation of energy during these utterances, but it is directed more strongly on the vocal (including respiratory) organs, to those organs which serve especially during emotions. Strong emotion tends to more automatic, inferior utterance.

I suppose, then, that strong emotion, or rather, of course, the nervous discharges accompanying it, may have led in the becoming speechless man to so rapid a discharge of so many nervous arrangements of higher cerebral centres, in this case for words, that in the great struggle resulting, the survival of the fittest was a survival, not of the best for the then external circumstances, but of but one word, or of some syllable or syllables: the fittest was then an interjection, or only some parts of sentences or parts of words fused into jargon.

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[1] Probably there is a more complex condition; during strong emotion, there are rapid and strong discharges on the vital organs which soon lead to exhaustion of these organs, thus there will be a multiplication of effects; the cerebrum, especially its highest centres, will be ill served with blood.

Rapidity of emotion is the great thing in the above considerations. In another way we are concerned with rapidity—with rapidity of the being taken ill, which means rapidity of destruction of nervous arrangements in the left half of the brain.

In all cases of nervous disease we must endeavour to estimate most carefully the element of rapidity of lesions, not only the quantity of nervous elements destroyed, but the rapidity of their destruction. We have to try to estimate the momentum of lesions. We, however, use the term "gravity of lesions." I do not pretend that I can show the bearing of the factor rapidity on symptomatic conditions in cases of affections of speech. It is fair, however, to consider it in this regard to suggest a basis for future investigation. The slowness with which a lesion comes on is determined by the slow onset of the symptomatic conditions; a small sudden hæmorrhage produces greater, but a more temporary effect, provided it does not at once kill, than a large, slowly developed softening. But in the latter the paralysis or other defect depending on mere destruction of the centre is more local and more lasting.

A very sudden grave lesion would render unavailing all external circumstances, and would prevent all emotion, for it would produce unconsciousness at once; a less grave one would prevent the influence of external circumstances, but would develop emotion. A lesion of very little gravity would allow external circumstances to act, but to act quietly and allow clear propositions to be framed. Repeating in effect what has been said, it does not follow that the lesion in the latter case is not an extensive one; the question we are now about is as to the gravity of lesions, metaphorically speaking mv of lesions.

We should then note whether the aphasia and hemiplegia are rapidly produced, whether or not there be sudden loss of consciousness, and note also the length of the coma, if there be any. Everybody does this for clinical purposes, in order to obtain empirical evidence of the kind of pathological process, whether it be clot, softening, &c.; but we should use the facts also as regards the interpretation of the kind of recurring utterance the patient may have. Perhaps the bearing of rapidity of destruction on the nature of recurring utterances is not made evident in the above. It is not here *directly* a question of rapidity of destruction of any centre, or of "shocks" given to centres connected by fibres with the one suddenly damaged, but a question of the indirect effect of rapidity of destruction, of such destruction in so far as it is a *sudden removal of control* (or inhibition) over lower centres. Of course by "lower centre" we do not mean one geographically lower, but one anatomically and physiologically lower. The destruction can only directly *cause* negative symptoms; it neither directly causes the utterance nor interferes with it; it may be popularly said to cause it and to affect it indirectly, but it is better to say it permits it. In so far as cases of aphasia are concerned, the above is hypothetical. Let us consider the principle stated more generally. In other cases of nervous disease rapidity of dissolution is, I think, evidently an important factor with regard to symptomatic conditions. The epileptic maniac is the most furious of all maniacs. Why most *furious*? The fury is the psychical side of what is physically, of course, great activity of nervous arrangements, it is activity of lower nervous arrangements; for as the furious maniac is insensible also, it is plain that his highest nervous arrangements are *hors de combat*. Why, then, are the lower nervous arrangements so active? I believe it to be because the process of dissolution effected in the epileptic paroxysm prior to the mania is the most rapid of all processes of dissolution—control is most rapidly removed. Let me try again to make this clear by formulating it otherwise. We have not only to consider "depth" of dissolution, but the rapidity with which the dissolution is effected—control removed. I believe we may say that the deeper the dissolution, the more general and more automatic are the processes remaining, and that the more rapid the dissolution the more excitable are the nervous arrangements of those processes So that with shallow and yet rapid dissolution the actions are elaborate and also busy; with deep and rapid dissolution, they are simple and also violent (it is not a question of "importance" of actions).

I presume, then, that the more rapid the destruction of the part in the left half of the brain, the more excitable would be the nervous arrangements in the corresponding undamaged right half, and thus that the activity of it would be greater—greater for some time after the onset of the illness. But the more rapid the destruction, the more quickly there would be loss of consciousness; thus the then external circumstance would be unable to act at all, and although the lower centre might be more excitable than usual, there would be nothing for it to manifest as a result of what was saying or doing at the onsei illness.

To recapitulate. By considering (1) the external circumsta the time of the being taken ill; (2) the intensity of the emotior under which the last attempt at speech was made; and (3) the of the lesion, we may, perhaps, be able to show why this or that recurring utterance remains in particular cases of speechlessn

We shall now consider phenomena analogous to recurring ut in some cases of defect of speech (No. 1, p. 115), in order to wi basis of investigation.

The patient who has defect of speech may get out a word, wrong, and go on uttering it; or he may even get out a prope such as "Very well," and go on uttering that in rejoinder to questions to which it is irrelevant, being aware of that irrelevan use Gairdner's expression, who drew attention to this peculia patient gets the word or phrase on his "barrel-organ." It a temporary recurring utterance; the permanent recurring utte loss of speech may be called permanent barrel-organism. In defect of speech the patient may write a little, and then go on the same syllable or word or phrase over and over again. Here is evidence, although indirect, of "barrel-organism" of w syllables.

The following is a letter written by a patient who had d speech: it was not punctuated. He first wrote his address c which for obvious reasons I do not give, I substitute "Hur North Newington Road" for it; the address recurs in the let factitious address is put in place of it:—

"Dear Sir,—I feel very well just now" ("for now" and " came next, but are crossed out) "for Hurst Row I feel very now for thingg in the first way for the thamk now. I know no in the first now in the first now in the Newington Road. I first way in Newington Road the poor way is the best way for n best way for me is the best way for me is the best way the wa best for me for ways kept for me for kept ways kept me for v for me."

Here ends the letter. There is, however, plainly somethi than barrel-organism in it.

From what would be generally called the physical sphere w illustrations in some cases of *loss* of speech of "barrel-organis after getting an aphasic to protrude his tongue, we ask him to hand on his head, he may, instead, open the mouth and perhaps put out the tongue. This, I grant, may be of doubtful interpretation.

Here let us note another peculiarity in cases of defect of speech, which, perhaps, can only be shown to concern us indirectly in regard to what has been said. It is a very important matter in the elucidation of the nature of defects of speech, and may properly be mentioned now in order to suggest inquiry if there be anything analogous in the recurring utterance in cases of loss of speech. In some cases of defect of speech we find that the patient who has uttered a word or phrase correctly, and who goes on re-uttering it as a reply, cannot repeat it when he is told and when he tries. He has said correctly, "I don't know." We tell him to say "I don't know"; he fails. Whether there is anything strictly analogous to this in the case of permanent barrel-organism Nos. 1, 2, and 3, as there is of "no," No. 4, will not be easy to determine. For if we ask the patient to *say* his elaborate recurring utterance, just as we ask patients who reply "no" to say that word, there is the difficulty that the recurring utterance comes out when they *try* to say anything; and thus its coming out may be not saying it, but simply uttering it as at any other time. I confess I have no personal observation on this matter worth mentioning. It has long been known that some aphasics at least cannot say parts of their recurring utterances. Thus a patient of Bazire's uttered "sapon, sapon," but could never say "sap" or "pon" only. Trousseau makes analogous observations. But here obviously there is a difficulty. What we call part of a word is really another word or syllable. A patient who consulted me for loss of speech learned to *say*—not merely to utter—"Battersea"; but she could not say "batter." When asked to say it, she said "Battersea."

Here we may make further remarks on dual mental action, taking this time not the process ending in ideation or perception, or both, but the process ending in voluntary, as distinguished from automatic actions. What we now say may serve to integrate remarks which necessarily came now and then incidentally in discussing different parts of our main topic. We make a few preliminary observations.

We must bear in mind that "will," "memory" and "emotion," are only the names men have invented for different aspects of the ever present and yet always changing latest and highest mental states, which in their totality constitute what we call consciousness—consciousness being really a name for a series of varying and different consciousnesses. There is, however, a double series, subjective and objective consciousnesses. Each one of the series of object consciousness is secondary to a state of consciousness or subconsciousness, representing us altogether, subject-consciousness. It is this duplication, I suppose, which gives us the feeling that we have a sort of general and persisting consciousness, and are also now, now, and now, conscious, this or that particular way, and which makes us say that this or that sensation or image comes *into* consciousness, or that we have a sensation and know that we have it.

Taking ".will," "memory," and "emotion" to be real independent entities or faculties reminds one of the old woman's remark, "How lucky it was that Adam called all the animals by their right names."

To say a word or proposition when told "for the sake of saying," is not a language process at all. A speechless man's inability to say "no" when told is a thing of the same order as his inability to protrude the tongue when told. The consideration of such facts will help us to classify the phenomena of cases of aphasia on a deeper basis than that of language. To use an expression somewhat loose in this connexion, there is loss of certain voluntary actions in some cases of aphasia, with conservation of the more automatic—a dissolution affecting more than language processes, and affecting language processes not so much as language processes, but as they are some of the voluntary actions. We have to consider speech on this wider basis in order that we may be better able to see how speech is part of mind, and thus to get rid of the feeling that there is an abrupt and constant separation into mind *and* speech.

It is in considering the nature of these phenomena that we see, I think, the bearing of Spencer's remark on the distinction betwixt voluntary and involuntary operations. Where we have to do with quality of mental processes. In the voluntary operation there is preconception; the operation is nascently done before it is actually done, there is a "dream" of an operation as formerly doing before the operation; there is dual action. Before I put out my arm voluntarily I must have a "dream" of the hand as being already put out. So too, before I can *think* of now putting it out I must have a like "dream," for the difference betwixt thinking of now doing and now actually doing is, like the difference betwixt internal speech and external speech, only one of degree; in one there is slight discharge of a certain series of nervous arrangements, in the other strong discharge of that series. The "dream" must occur before I either think of now doing something or before I actually do that thing, just as words must be reproduced in me before either I can say them to myself or aloud. To say that we know what we are going to do, or that we are intending to do this or

that amounts to admitting the above hypothesis. These expressions imply our having a nascent excitation of nervous arrangements representing the parts formerly concerned in doing that which is now to be done again. If we say we are trying to do something, we mean similarly, and so we do if we say we remember how to do anything; there being no "faculty" of remembering an action apart from having the action (as it was formerly doing) again actual, although faintly actual, nascent. To "will," to "know," to "intend," to "try," to "remember," have each, in their several contexts above, the same meaning. They are different names for the subjective reproduction which precedes objective reproduction; they are names for "[illegible]reams" of what was once doing and is now to be attempted aga[illegible]eed the simple expressions, "He speaks," "He does so [illegible]ply the duality; imply objective activity following subje[illegible]y. "He speaks," or "He does," implies a then tempora[illegible]ication of a person into subjective and objective, "he" repres[illegible] that person's whole self of course, and "speaks" or "does," represents something doing by his whole self. "He" represents the whole of him in the stage we have figuratively called having a "dream" of a past operation, "speaks" or "does" represents the whole of him in the stage of imitating it. (We say figuratively, because we do not mean a visual dream, but having again sensations representing parts of the body
[illegible]d as being moved thus or thus.) To take a simpler case,
[illegible]hich, however, usage will not allow application of the
[illegible]nd "involuntary," "he has this sensation." Sub-
[illegible]nsation arisen in him which associated directly
[illegible] indirectly with all others—all that have been
[illegible]ere is disassociati[illegible]e sensation is considered
[illegible] that are or[illegible]in relation to those
[illegible] fanci[illegible]n thus: "*He*
[illegible]t is a rhythm
[illegible]hat a speech-
[illegible]ming that he
[illegible]

themselves, their nervous arrangements being broken up; he has lost a part of himself. And were we to say that the patient desires to do those things, we should only mean that he retains the subjective actions, which in health are precedent, and are to him the preconception of the objective actions which cannot follow, for the simple reason that they do not exist. I suppose such expressions of duplication of mental states, "ideas *of* actions," "memory *of* words," "feeling *a* sensation," and "*knowing that we have* this or that feeling," &c., imply that there is first and subconsciously the action, or the word, or the particular sensation associated with (because arisen out of) the already organized in our whole selves (subjective actions, words, &c.), before the second reprodu[illegible] them or rather of their duplicates in relation to what is then or[illegible]m the environment. A subjective word is first a centre of [illegible] with all other organized words, symbolizing all other image[illegible]fterwards a symbol of a particular image; an objective wor[illegible] first a symbol of a thing and next acquires new associations with other words, then using along with it and similarly arisen to indicate new relations of things in the environment.

In healthy speech it is hard to see how we can have a proposition (either in the degree of internal or in that of external speech) before the words of that proposition have been subjectively revived. Otherwise, how should we be more than machines? Words must have arisen in us in an order symbolizing states arisen in us, before a [illegible] we utter can have, *for us*, any meaning as symbolizing [illegible] of things one to another in the environment. To r[illegible] we must first remember the words and then say [illegible]

We do not, as already incidentally implied, n[illegible] subjective and of the ob[illegible]e process are arr[illegible]

Let us return to [illegible]movement of [illegible] here begins, o[illegible]h cor[illegible] of some opera[illegible] If I am to pu[illegible] *with* the han[illegible] ositions [illegible] in the limb engaged, and (b) currents coming from the rest of the body, as being thus or thus fixed, or as thus or thus variously displaced during the particular movement of the limb. And of course there is an accompanying series of outgoing currents, for if there were no motor element we should have no knowledge of the relations of the parts moved to one another and to the rest of the body; incoming currents would not suffice to give this relation any more than incoming currents from retired retinal elements, without accompanying ocular movement, would suffice to give us a knowledge of the relations of these elements one to another; and, plainly, unless retinal elements are first known as out of one another, impression on them by objects would give us no notion of the exterior extension of those objects. Similarly, until we have already a knowledge of the relation of parts of the body one to another, we could not begin to make any movement of part of the body to operate on the environment. During discharge of this unit a psychical state of what was formerly doing arises. When the operation is to be repeated, to be done now voluntarily, the other unit discharges the operation, starting from an attitude of the body and passing on to the end of the operation by the limb. Manifestly this must begin by an attitude of the body, and the progress in the arm will be from the shoulder to the hand, in an opposite order to the "dream-movement." It is during the play of these two movements that what is called muscular sense arises. The psychical side of one process is part of what we call desire, of the other of what we call effort.

That subjective states arise in an order the opposite of that they are arranged in in corresponding objective states, is supported by the analogy of some dreams. A noise develops a dream, but, sometimes at least, the noise, which in reality acts on the sleeper first, is last in the dream it excites. In all dreams provoked by local excitations I suppose the transfigured excitation comes either last or later than the excitation itself. And I suppose that in such a proposition as "gold is yellow," the subjective order is the reverse of the sequent objective order; for our concern is first with the yellowness of gold, not with gold. This view is, I think, in harmony with what was said on transposition of syllables in mistakes of the slightly aphasic and the healthy. I believe there to be a duality also in automatic operations; but in this case the movement has so often followed the dream that the two are nearly equally perfect and easy. The voluntary operation, the prior "dream," is imperfect, or, if perfect, the movement done after it does not imitate it closely; we try to do so, and fail. Becoming automatic by repetition is, on the physical side, for the two units to discharge more nearly together, because lines of less resistance are established; there is less delay betwixt them, and thus preconception through the discharge of the first unit and less sense of effort during discharge of the second. Thus less and less consciousness attends processes the more they become automatic; subjective and objective actions become, as it were, nearly fused.

Let us now take other kinds of nervous affection which show something analogous to permanent barrel-organism of speechless patients. We wish to point out that there is evidence that operations going on at the time of unconsciousness supervening remain nascent or in abeyance, are not always swept away, during the unconsciousness. During the restoration to consciousness they become active again; on full restoration to consciousness, they cease.

Directly after or in coma from various causes, we occasionally see a reversion to actions doing], when the comatosing lesion occurred. An ostler coming round from coma, due to the kick of a horse he was grooming, began to "hiss" as grooms do when engaged in rubbing down horses (Brodie).[1]

Abercrombie writes: "A lady, whose case has been communicated to me, was seized with an apoplectic attack while engaged at cards. The seizure took place on a Thursday evening, she lay in a state of stupor on Friday and Saturday, and recovered her consciousness rather suddenly on Sunday. The first words she then uttered were by asking, "What is trumps?"

I suppose the above instances show that actions nascent at the time when the illness occurred remained so during the coma, and went on again actively when consciousness was being restored; on full restoration to consciousness they ceased, the barrel-organism was temporary, because the highest nervous centres were only temporarily *hors de combat*.

The following case, kindly supplied to me by Dr. Buzzard, has never been published. It may be taken as a faithful record, since the observer's qualifications as a neuropathologist are of the very highest. It is one of extreme value in many ways.

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[1] I suppose the following to be analogous, although consciousness was only partly restored; the action of laying the oilcloth had become "barrel-organic" A woman I was asked to see in a surgical ward of the London Hospital, who fractured her skull by a fall when laying down the oilcloth on a staircase, kept, during partial unconsciousness a few hours before death, manipulating the counterpane of her bed. We supposed this to be a continuance of the action of laying the oilcloth, the patient desisted for a time, at least when the nurse assured her "it was properly laid."

"About fifteen years ago, a medical friend of mine, some 57 years of age, was pitched on to his head, owing to the horse which he was riding having put his foot in a drain. He was taken home in a state of profound insensibility, in which I found him very shortly afterwards. From the symptoms it was probable that he had fractured the base of his skull. For some hours (I have no note of the number) he lay in a state of deep coma, incapable of being aroused. From this his condition gradually changed to a state in which he appeared to sleep, but would reply "yes" or "no" to questions put to him. He did not appear to notice anything about him, and never volunteered a remark, but scarcely ever failed to reply in the way described, and with such an amount of propriety that I remembered it was difficult at first to believe that he was unconscious. Offered food, he would sit up in bed, drink it without a word, lie down again and go to sleep. On one occasion I wanted him to take a common black draught, but he appeared to resent this, said "No! no!" fiercely, and turned away. By a little persistence, however, he was induced to swallow it. He would get out of bed, take the chamber utensil out of its place of concealment, and pass urine regardless of anyone in the room.

"One morning, to my astonishment, he got up, walked to his shaving-glass at the window, and proceeded to lather his chin. He then shaved himself, with very fair completeness, and returned to bed. I happen to know that this was the way in which he invariably commenced his morning toilette, for he was a late riser, and he had frequently risen and shaved himself during my visit. From this day he got up every morning about the same time, repeated the operation of shaving, and then returned to bed. The action was performed with the same evident unconsciousness of the presence of others which we see in the somnambulist. Having returned to bed he would lie for the rest of the day in a dozing state, which was only interrupted by his taking food and passing his evacuations, which actions were evidently performed in a purely automatic manner.

"Upwards of a fortnight from the time of his accident he woke up into consciousness, noticed persons in the room, and made some slight remark. Little by little during the next hour or two he began to converse at greater length, referred to circumstances relating to his business which had occurred before his injury, and it became presently evident that the time during which he had been in bed had left no trace upon his memory. Of the injury itself he knew nothing whatever, the last thing which he remembered being that he was riding on horseback.

"When asked the day of the week and month he unhesitatingly gave the date upon which (more than a fortnight previously) he had met with the injury, and became quite angry when assured that he was mistaken. The period of his illness had been cut out, as it were, of his life in the cleanest and most complete manner, and not a trace of it has ever since presented itself to his mind."

In the above-mentioned cases the patients recovered, and thus their cases are not closely analogous to our cases of permanent barrel-organ utterances in aphasia. A patient who recovers soon from aphasia loses his recurring utterance, becomes able not to utter it. These cases are analogous, and so is the temporary barrel-organism of some patients with defect of speech. In the permanent barrel-organism or recurring utterances of patients who remain speechless, the restoration of the part (in the left half of the brain) placed *hors de combat* is impossible; it was destroyed altogether. The following case is more closely analogous to these cases than the above. Abercrombie relates (after Conolly): "A young clergyman, when on the point of being married, suffered an injury of the head by which his understanding was entirely and permanently deranged. He lived in this condition till the age of 80, and to the last talked of nothing but his approaching wedding, and expressed impatience of the arrival of the happy day."

What does recovery in the case of the cures of barrel-organ actions mentioned mean? It means restoration to functions of some or enough of the highest nervous arrangements; it means return of control. But in cases of speechlessness from entire destruction of nervous elements there can be no restoration; the control does not return. The recurring utterance remains, and by repetition becomes highly organized.

WORDS AND OTHER SYMBOLS IN MENTATION.

[*Medical Press and Circular*, August 30, 1898, vol. ii, p. 205.]

WHEN anyone says that words are essential for thought, he may, I suppose, mean that words or *some other symbols* (or both words and other symbols) are so. Pantomimic actions, for example, are symbols analogous to words) and untrained deaf-mutes use these, not only for inter-communication, but also in thought. There are in all men, I submit, actions not reaching the rank of what would commonly be called pantomime, which are symbols in mentation. In all cases it is meant that propositions of symbols (verbal or pantomimic) serve in mentation, evidently to speak is to propositionize. This being said, I may, without misleading, speak of symbols as if they were independent of one another. I use the term "action" for the psychical "side" of what are physically movements.[1]

Since lower animals think, it may be asked, "What are their symbols?" if symbols are necessary in mentation. They have no words, but I submit that they have other symbols—inferior symbols, symbols but little symbolic—which serve in their lowly mentation. This matter I can only deal with after speaking of symbols in the case of man I begin with words.

A word is a psychical thing, but of course there is a physical process correlative with it. I submit that this physical process is a discharge of cerebral nervous arrangements representing articulatory muscles in a particular movement, or, if there be several syllables, in a series of particular movements.

Strictly, no doubt, the physical basis of a word, or rather syllable, is a sensori-motor, an audito-articulatory, nervous arrangement. But in speech, the objective part of verbalizing, our main concern is with the motor element of the physical basis of words. It is current doctrine that the part of the brain destroyed in cases of loss of speech (aphasia) is motor, and that it is a part of cortex cerebri (Broca's region) representative of movements of the tongue, palate, lips, &c. Artificially induced discharge of what in the monkey is homologous with Broca's region, produces movements of the tongue, palate, lips, &c.; and so far as we can see, there are no other effects from that discharge. And I suppose no one doubts that very strong discharges of Broca's region in man (such discharges, I mean, as occur in cases of "cortical epilepsy") produce convulsion of the muscles of the same parts; this convulsion is nothing other than a "running up" of many movements of the muscles of the parts mentioned into a stiff struggle. Dr. Charlton Bastian, however, thinks that the whole of the so-called motor region (Rolandic area of the cortex cerebri) is not motor but kinæsthetic. The opinions of this distinguished physician deserve most respectful consideration. Possibly, nearly all I say about what I call the motor part of the physical basis of words, might be accounted for on Bastian's doctrine of kinæsthesis. I continue, however, to speak of Broca's region as motor.

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[1] There are, I think, also arbitrary image symbols serving in mentation; of this matter I shall speak later.

It has been said that cases of aphasia, let us suppose cases of speechlessness, give proof that thought is possible without words. We do not often encounter a case of complete speechlessness; the patient whom we call speechless can nearly always reply by "yes" and "no," and these words so used are, or are in effect, propositions. I shall suppose a case in which a patient can utter these two words, and yet cannot use them, cannot reply with them; he is then completely speechless. I have long ago put forward the opinion that the speechless man is not wordless. There is disease of but one half (left) of his brain, and I submit that there are nervous arrangements, the physical bases of words, in the fellow, undamaged (right) half. Using again the term "verbalizing" to include all services of words, there are at least two ways in which words serve; they serve not only in speaking, but in the reception of the speech of others. In a case of aphasia with word-deafness (a special imperception) there is a loss of receptivity of words, but this is, I submit, because, speaking now of the physical, the nervous arrangements for articulatory movements in the right half of the brain cannot be reached. I am supposing a case of speechlessness without that complication; in this case the patient understands what we say to him, on simple matters at any rate, he receives our speech, has that services of words.[1] Further, the damage to the left cerebral hemisphere

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I now return to the dictum that the physical basis of a word is an audito-articulatory nervous arrangement, and as I am here dealing with speech and not with other services of words, I consider only the motor part of that basis.

When a man says aloud, "Gold is yellow," or says that to himself, there is correlative with the psychical things (the words of this proposition) discharge of cerebral nervous arrangements for four movements of his articulatory muscles. At the risk of too great iteration, I remark that it is not said that a word is an articulatory movement or discharge of cerebral nervous arrangements for one. The notion that movements or cerebral nervous arrangements for them serve in mentation is grotesque. The statement is that a word, a psychical thing, is concomitant with a movement of the articulatory muscles—properly that it is concomitant with discharges of certain cerebral nervous arrangements representing one or more articulatory movements—and that words serve in mentation. I will remark in another way on this distinction.

We must not say that speaking aloud is a physical process only, and that speaking to oneself is a psychical process only. There is a double process in each. When speaking to oneself, there is a slight discharge of motor cerebral nervous arrangements representing movements of the articulatory muscles, with a correlative faint psychical state; in this case the discharge is so slight that lower centres are not engaged and no articulatory movements are produced. In speaking aloud there is a strong discharge of the same nervous arrangements with a correlative vivid psychical state—in this case the discharge is so strong that lower motor centres are engaged, and finally there are movements of the articulatory muscles in a particular sequence. In a severe case of bulbar paralysis the patient cannot articulate, but he can speak "internally," and can write out what he says "internally." The cerebral motor nervous arrangements of Broca's region continue in the case of bulbar paralysis to represent articulatory movements, although the articulatory muscles and their immediate (medulla) centres have disappeared (atrophy); these cerebral motor nervous arrangements have, as they have in healthy men, a psychical "side."

We must not say that, whilst speaking aloud ("external speech") is an objective process, speaking to oneself ("internal speech") is a subjective process; both are objective, there are two degrees of objectivization. And as to images which words symbolize, both ideation and perception are objective processes; they are different degrees of objectivization.

To return to movements and to actions, the supposed psychical "sides" of movements. It may be said that in the case of other movements—I limit attention to those of conduct—there is no psychical "side" to energizing of the motor cerebral nervous arrangements representing them, that, for example, when a man moves his arm there is a physical process only. Yet if it be true that with discharges of cerebral nervous arrangements producing certain movements of the articulatory muscles there are concomitant psychical states, words, it would be remarkable if there were not also psychical states, actions, accompanying discharges of the cerebral motor nervous arrangements which produce other movements (of conduct). Disregarding this, I consider the matter anew. There is, I submit, proof that discharges of cerebral motor nervous arrangements for some movements other than those of the articulatory muscles, have a psychical "side."

When a man's arm is cut off he has a spectral hand. In most cases he can "move" his spectral hand, may try to pick up things with it, can put its fingers in positions he imitates for us with the intact fellow member. I submit that the movement as I called them of the spectral hand are really actions, that is that they are psychical states, concomitant with activities of motor cerebral arrangements which continue ignorantly to represent movements of the lost hand, if I may use that expression.[1] We cannot suppose that the man had not a spectral hand before amputation of his arm, and that it appeared only after the operation to remind him of his former entire condition. I submit that all entire men have not only movements of their hands, but also actions—psychical states—correlative with activities of the cerebral nervous arrangements effecting these movements. When a man's arm is cut off, he finds this out.[1]

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[1] The amputated man is physically something in the same case as is a man who has complete atrophy of all the muscles of his arm (Duchenne-Aran type of progressive muscular atrophy). I say something, because in the former case the anterior horns are intact, in the latter absent (wasted). And I should imagine that this would make a difference in the definiteness of spectral movements—expecting the movements, properly actions, of the spectral hand in the former case to be more definite than those in the latter. I do not know that there is that difference in the two cases. I do not, indeed, know whether or not there are

To repeat *mutatis mutandis* what was said on differences between speaking aloud and speaking to oneself.

When I actually move my hand there is a vivid psychical state or action concomitant with strong activities of motor cerebral arrangements representing the movement; when I think of moving it there is a faint psychical state, or action, concomitant with slight activities of the same nervous arrangements. To take another case. When a deaf-mute is communicating with his fellows, there are strong discharges of cerebral motor nervous arrangements with accompanying vivid psychical states, actions; when he is thinking there are slight discharges of those nervous arrangements, and their accompanying psychical states, actions (so-called "ideas of movements"), are faint; in the former case there

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spectral movements in the case of progressive muscular atrophy I think it likely that there are, because words arise during activities of cerebral nervous arrangements representing absent muscles in the other case of progressive muscular atrophy, that called bulbar paralysis. It would be very difficult to make some patients understand what was meant by asking them if they felt *as if* they could "move" a hand which is evidently useless for any degree of actual movement. The question is one of extreme importance in the anatomy and physiology of the nervous system as they correspond to psychology. The hypothesis is that degrees of definiteness of "projection" (see p. 184) are correlative with degrees of involvement of the motor centres of the cerebral sub-system, from the highest downwards; in detail the supposition is, to illustrate by movements of the arm that, on amputation of this limb, there is most definite "projection"; that in cases of atrophy of anterior horns for the arm, there is less definite "projection," and that there is still less definite "projection" in cases of destruction of the so-called arm-area of the Rolandic region of the cortex. This, as said, is hypothetical, it is only suggested as a method of investigation. "In some cases of impotence of limbs from other causes, there are spectral movements; the other day a hospital patient told to move her toes, tried and could not; she volunteered that she felt as if she did move them when she tried."

[1] I do not mean that these actions, psychical things, are "ideas of movements"; we have, indeed, no ideas of any parts of our bodies. To take another kind of case: a colour is not an idea of activity of any sensory cerebral elements representing the retina; colour is a psychical thing, a sensation, concomitant with the physical thing, activity of certain cerebral sensory elements. Similarly, we have no ideas either of muscular movements or of activities of cerebral motor nervous arrangements representing them, there are actions, things psychical, concomitant with energizing of those nervous arrangements. I cannot but think it important to bear this distinction in mind. Taking another case, it would surely be erroneous to speak of subject-consciousness (self-consciousness or self) as being a consciousness of the whole organism or of the highest centres. No doubt, a very different thing, subject-consciousness is correlative with activities of nervous arrangements representing all parts of the body as one whole.

are actual movements of muscles, in the latter the discharges are too slight to produce actual movements. Suppose the deaf-mute were to lose both arms, he would try to move them, and would then, I suppose, have for himself vivid spectral pantomime during energizing of cerebral nervous arrangements representing the absent muscles of the limbs; the pantomimic actions arising during slighter discharges of those nervous arrangements, would serve in his mentation the same as when he was entire.

I next state an hypothesis as to the evolution of pantomime, beginning with the physical.

I suppose that movements during pantomimic actions (I now limit attention to the pantomime of ordinary men, and do not speak of that of deaf-mutes) have arisen out of what I may call the ordinary movements of conduct. To take a case. I suppose that beckoning (the movement used when signifying that someone is to approach) has arisen out of the ordinary movement, or, I will say, operation, of pulling somebody towards oneself. The operation of pulling was once done, and at length some small and modified part of it comes to stand for that operation, for that out of which it has arisen stands for the movement of pulling someone towards oneself. More generally, some parts of operations come to stand for whole operations.

Of course, when dealing with the movements just mentioned there is, as in other cases, tacit reference to cerebral motor nervous arrangements representing them. As the movement of beckoning arises out of, and comes to stand for, the operation of pulling towards, there must be an evolution of new motor nervous arrangements out of those motor nervous arrangements, the activity of which produces the operation itself. There is, in the cerebral area concerned, more or less detachment and independence of the new nervous arrangements *standing for* the operation from those effecting the operation itself. So far I have spoken of the physical.

It was contended that movements of conduct, for an example the operation of pulling, have a psychical side, and, if so, then, *pari passu* with the evolution of the movement standing for an operation there comes an attendant psychical state, a pantomimic action—a symbol—is the psychical side of energizing of the new nervous arrangements which have been evolved out of those representing the movement of what I called the operation.

When these new nervous arrangements are strongly discharged there are actual movements, and there are concomitant vivid psychical states, when they are slightly discharged there are concomitant faint psychical states and no actual movements.[1]

When I arrange books on a table there are visual and tactual images, and there are actions. Physically there are imprints on the retinæ and on finger-tips and there are movements of the arm (representing cerebral nervous arrangements understood). Now suppose I think of arranging the books. I have faint visual and tactual ideas, and there are faint actions, and physically there are slight central, cerebral discharges, the peripheral parts not being engaged. In this case the actions are scarcely symbolic, and yet probably to some extent so; so far, at least, as they are not mere repetitions of the former actions, but incomplete and modified reproductions of them. In rudimentary symbols of this class I consider some lower animals think—in symbols the very low homologues of what in man are pantomime—in symbols but little symbolic.

Of course, the separation between pantomimic actions and such actions spoken of in the illustration given (arranging books) is arbitrary; very likely there are degrees from actions simply done over again, not really symbolic, through actions a little detached from those concomitant with the ordinary movements (actions but little symbolic) to those actions so much detached as to be symbols having the rank of pantomime.

It sounds grotesque to say that manipulating and certain other gross operations, or rather the correlative actions, are reasoning, but I think there is reasoning in its lowest form—or that there is at least that out of which what is called reasoning has been gradually evolved. To return to the former illustration. The action during actually arranging books is reasoning in its lowest form; the action when thinking of arranging them is a next higher grade; a purely symbolic arrangement of them in thought, by actions concomitant with activity of new nervous arrangements representing modified parts of the operations, is a sort of reasoning. Certainly when words come to be symbols of the actions (symbols of symbols) in the case supposed there is what is commonly called reasoning.

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[1] If this hypothesis be valid, the genesis of words (strictly, of course, the genesis of cerebral nervous arrangements which are the physical bases of words) is accounted for. The nervous arrangements of "Broca's region" represent movements of the muscles of the tongue, palate, and lips. This region represents also (Krause, Horsley, and Semon) movements of the larynx and of the pharynx. It is concerned with the "commonplace" movements of eating, swallowing, &c. I suppose that the motor nervous arrangements of Broca's region which are correlative with what are psychically words have arisen out of those for the "commonplace" movements of the tongue, palate, and lips, and have become greatly or completely detached from the latter, and then serve, not for their own ends, but stand for operations. They do not, however, stand for the operations out of which they have arisen, but they, the correlative actions, become more widely symbolic. If so, Broca's region in man differs from the homologous part of a dog in there being in it more specialized, new, nervous arrangements in addition to what we may call the ordinary or old ones.

Words in themselves have no meaning; they are but symbols; it would matter nothing whether a certain small quadruped were called "cat" or "tac"; either would do as a symbol for the image we have when we think of a particular cat. Again, words only come to have speech value when they are in propositions; to speak is to propositionize, it is not merely to utter words.[1] (Similarly, *mutatis mutandis* for pantomimic actions and pantomimic propositions.) Of course, a single word may be, in effect, a proposition, other words completing the proposition being understood, internally revived. Passing over the obvious cases of "yes" and "no," if a father calls to his son "Here," there is in effect the proposition "Come here." Hence speaking is symbolizing a mental operation.

I do not mean that propositionizing occurs only when we speak to tell others what we think, but that it occurs when, so to say, we are telling ourselves what we think. Whether "gold is yellow" is said aloud or whether it is thought that gold is yellow, there is propositionizing, a relating of images to one another in a particular way. But it may well be said that whilst we may declare that there are images of this or that yellow object, there can be no image of yellow in, for example, the proposition just instanced. We have seen this or that yellow object, but no one has seen, and thus can have no image of "yellowness." If so, it may appear that the word "yellow" in the proposition instanced has nothing to symbolize.

I suggest that there is a class of symbols differing in nature from those of words and pantomimic actions, which also serve in mentation. I used to call them "arbitrary perceptive signs," but now "arbitrary images." I submit that when we think "gold is yellow" the mental process symbolized by the proposition is, or rather that the first stage of the process is, thinking that a gold thing is a yellow thing. I imagine that each person has for his fancied general notion of yellowness a particular image of some particular yellow object, and that this image serves him when no particular yellow object is referred to.

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[1] To say a word when told to do so is not really a speech process. An aphasic may be able to reply "no" when he cannot say "no" when told. (Strictly to "*say* a word when told" is not a correct expression.)

[2] Strictly speaking, internally or externally, and symbolizing generally, is a relating of images to one another, *and is thus an integral part of mentation.*

Obviously, this is not all. This arbitrary image is associated with other, possibly with all other, images of yellow objects seen and remembered, and from their secondary excitation there is a fancied notion (or concept) of yellowness. The gold thing is grouped with yellow things (classified). The word "yellow" is a symbol of some particular yellow image which is itself symbolic in the sense stated. Of course, I do not mean that we thus get a notion of yellowness; there is no such thing to have a notion of.

I will give another illustration. How, it may be asked, can the word "triangle" in a proposition develop in us the image of a triangle of whose particular shape nothing is affirmed, when actual triangles are dissimilar, equilateral, isosceles, and scalene? I feel that I myself have an image of a particular triangle, viz., an equilateral triangle, which, I suppose, serves in thought for the fancied general notion of triangle. Suppose the following proposition is in question: "The three interior angles of a triangle are equal to two right angles." I cannot but think that everyone who heard and understood this statement would have an image of that kind of triangle, which had, in his experience, been most frequently associated with the word triangle, or of some triangular figure which had struck him. I call this an arbitrary image, because the image of any triangle would serve, and because the image serves intermediately as aforesaid. Suppose the above general statement is altered into the detailed one. "This is true of the equilateral, the isosceles and the scalene triangle." There would then be developed with each name an image of each kind of triangle, and I submit through intermediation of the arbitrary image. (Of course, I do not mean that a man has but one arbitrary image for the fancied notion of yellow; there may be many, they may change; so for all cases.)

IDEATION AND PERCEPTION: THEIR RESEMBLANCES AND DIFFERENCES.

I believe that ideation is commonly regarded as a subjective process; of course perception is recognized as an objective process. I submit that both ideation and perception are objective, two degrees of objectivization. The popular psychologist might aver that in perception there are real outer objects, things in themselves coloured, shaped, &c., things existing outside us and independently of us, and that in ideation there are "only ideas of those real objects." To take a simple case. When we see (perceive) what all agree to call a brick, there is not, as the popular psychologist supposes, a red-shaped thing outside us and existing so independently of us; the image, the red, square thing we then have, is part of our consciousness, is a ghost of ours. In ideation (thinking of the brick) there is a fainter image than that had in perception. In perception there is as much an "idea of a brick" as there is in ideation; properly the expression is that in each there is an image, the name of which is the word "brick." The idea is a faint percept, the percept is a vivid idea. The image had in ideation is a faint copy of that had in perception.

It is true that to the philosopher as well as to the peasant there *seems* to be in perception a red-shaped thing outside us; it is only a piece of knowledge to the former that there is not, but that there is simply a vivid image in himself. The faint image in ideation seems also to be something outside. I do not mean that it seems to be something present in front of us (it would in certain cases of brain disease even if we were blind). No one can think of what we call a brick except as something not self; there is then a faint image which is part of a remembered or constructed environment, seems in that way something outside.

In ideation the image is not only faint, but is indefinitely "projected"; in perception the image is not only vivid, but is definitely "projected." Of course, I do not mean by using the term "projected" that the image, part of our consciousness, is, or is also, something outside us; the term is only a device for expressing the "*seeming*" spoken of; extrusion of the image is not meant.[1]

Perhaps, in spite of what has been said, the rapid reader may, from the mere use of the word "image," infer[2] that it is inconsistently held that in perception there *is* a red-shaped thing outside us which, so to say, photographs its redness, shape, &c., on us, and thus gives us its redness shape, &c., and thus makes our image. There is no such contradiction. Images and all other states of object-consciousness come out of subject-consciousness, are not straightway produced by agencies of "real" outer objects upon us. It is not meant that in perception there is not something outside us, but that we have no knowledge of what that something is, except a belief in its existence. The *first* thing in that process the *end* of which is perception, is the action of this assumed external something, x, on us; in perception an image, a, is *forced out of us*, out of subject-consciousness; there is a revealing of self, consequent on the action of x. And of course, although one uses the expression "*the* image" this or that person's image is meant, what the external something can arouse in this or that person. In ideation there is a faint revival of the image had in perception, best considered when dealing with the correlative physical processes of the two degrees of objectivization.

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[1] There are degrees of *seeming* "exterioration" or "projection" of images (see footnote to spectral movements, p. 178); the degree of definiteness of this seeming is, I think, proportionate to the extent downwards of the discharge beginning in cerebral motor nervous arrangements. (No explanation is intended here.)

[2] He might infer this because in the article preceding this appendix I use the expression "imprints on the retinæ." I would be pedantic to do otherwise in a passing illustration.

The cerebral centres for ideation and perception are one and the same; during ideation there are only central (cerebral) activities, and these are slight; during perception the activities are periphero-cerebral and centro-peripheral, and are strong. In ideation there is a slight discharge of the same cerebral nervous arrangements which were strongly discharged during perception; correlative with this slight discharge is the faint image, the copy (not of a "real" outer object), a faint revival of the image had in perception. Ideation always precedes perception; perception does not always follow ideation.

We have spoken of two differences between ideation and perception: (1) Faintness and vividness of images; (2) indefinite and definite "projection" of these images. There is another difference, (3) in ideation the faint image is associated with numerous formerly acquired faint images; in perception the vivid image is associated with fewer formerly acquired faint images and is associated with vivid images now experienced. Ideation and perception are objective processes differing in triply compound degree.

There is, I hope, no implication in the foregoing that "the mind combines or associates, &c.," images or any mental states; they come out of subject consciousness in this or that association, in this or that combination.

I hold that the images (illusions) in cases of insanity are objective. An insane man's "illusions" are his perceptions; his "delusions" are his beliefs; most generally his symptoms are, or are samples of, his mentation which is the lower homologue of his mentation when sane. Whether a sane person sees a man in the corner of his room, or dreams that a man is in that corner, his mental state is objective. Were he to become insane—I will, however, take a real case: A girl under my care for intracranial tumour, became quite blind (quite deaf too), and later insane. One of her illusions (or perhaps I should here say hallucinations)

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1866.

[7] "Clinical Remarks on Emotional and Intellectual Language in some Cases of Disease of the Nervous System," *Lancet*, 1866, vol. i, p. 174.

(Report of a visit with Dr. Hughlings Jackson to the London Hospital. Demonstration of a case of right hemiplegia and aphasia. The integrity of emotional language as shown by the patient's ability to swear, and the destruction of his intellectual language, the loss of power to form a proposition, were clearly demonstrated.)

(Reprinted on p. 43.)

[8] "Notes on the Physiology and Pathology of Language," *Med Times and Gaz.*, 1866, vol. i, p. 659.

(Reprinted on p. 48.)

[9] "On a Case of Loss of Power of Expression," *Brit Med. Journ.*, 1866, vol. ii, pp. 92 and 326.

(Account of an epileptic who was convulsed on the left side without disturbance of speech and on the right side with speech defect. An interesting description of letters, copying, and a list of mistakes in speech of this patient

The second paper gives an admirable account of how such a patient copies, making no mistake, but never losing sight of his copy.)

[10] "A Case of Disease of the Left Side of the Brain involving the Corpus Striatum, &c. the Aphasia of Trousseau," *Lancet*, 1866, vol. ii, p. 605.

(Demonstration on a case (with autopsy) of loss of speech with right hemiplegia.)

[11] "Clinical Remarks on Cases of Temporary Loss of Speech and Power of Expression (Epileptic Aphemia? Aphasia? Aphrasia?) and on Epilepsies," *Med. Times and Gaz.*, 1866, vol. i, p. 442.

[12] "Remarks on Cases of Disease of the Nervous System in which Defect of Expression is the most striking Symptom," *Med. Times and Gaz.*, 1866, vol. i, p. 659.

[13] "Inability of Speechless Patients to execute certain Movements," (Addendum to paper), *Med. Times and Gaz.*, 1866, vol. i, p. 659.

[14] "Hemiplegia of the Left Side with Defect of Speech," *Med. Times and Gaz.*, 1866, vol. ii, p. 210.

1867.

[15] "Remarks on the Occasional Utterances of Speechless Patients," *Lancet*, July 20, 1867, vol. ii, p. 70.

(Report of a visit with Dr. Jackson to the wards of the London Hospital A short account is given of the patient whose stock utterance was "Jimmy." Some other similar cases are mentioned, and Dr. Jackson gave his views on the origin of jargon.)

1868.

[16] "Aphasia with Hemiplegia of the Left Side," *Lancet*, 1868, vol. i, pp. 316 and 457.

(This is the case in which Dr. Hughlings Jackson showed that loss of speech in left-handed persons was accompanied by left hemiplegia.)

[17] "On the Physiology of Language," *Med. Times and Gaz.*, 1868, vol. ii, p. 275, and *Med. Press and Circular*, 1868, vol. ii, p. 237.

(These are almost identical synopses of Dr. Hughlings Jackson's address before the Section on Biology of the British Association for the Advancement of Science, held at Norwich in July, 1868. Reprinted on p. 59. A much shorter report appeared in *Brit. Med. Journ.*, 1868, vol. ii, p. 259.)

[18] "Notes on the Physiology and Pathology of the Nervous System," *Med. Times and Gaz.*, 1868, vol. ii, p. 526.

(Reprinted on p. 65.)

1869.

[19] "Certain Points in the Study and Classification of Diseases of the Nervous System."

(The Goulstonian Lectures delivered before the Royal College of Physicians in 1869. These lectures were miserably reported in the *Brit. Med. Journ.*, 1869, vol. i, pp. 184, 210, and 236; and in the *Lancet*, 1869, vol. i, pp. 307, 344, and 379.

The second lecture was devoted to defects of speech. We reprint (p. 72) the account in the *Lancet*, which is just sufficient to show the intense interest this lecture must have had for us in the present day. But the views of the newly made Fellow of the College of Physicians were not considered of sufficient importance for more than the briefest report.

The Archives of the College of Physicians and the private papers of Dr. Jackson contain no report of these lectures.)

1870.

[20] "Speech Defects in Healthy Persons and Remarks on Utterances of Speechless Patients," *Brit. Med. Journ.*, 1870, vol. ii, pp. 459-460.

1871.

[21] "On a Case of Hemiplegia with Loss of Speech," *Med. Times and Gaz.*, 1871, vol. i, p. 703.

(Dr. Hughlings Jackson intervened in a discussion at the Clinical Society. The report is of no interest to-day.)

[22] "On Epileptiform Attacks commencing in the Right Hand," *Med. Times and Gaz.*, 1871, vol. ii, p. 767.

1872.

[23] "On a Case of Defect of Speech following on Right-sided Convulsion," *Lancet*, 1872, vol. i, p. 72.

(Report of a case referred to in the "Mirror" of the *Lancet*, March 18, 1871. Curious case of a young woman who had a seizure, from which she recovered entirely.)

1873.

[24] "On the Anatomical and Physiological Localization of Movements in the Brain," *Lancet*, 1873, vol. i, pp. 84 and 162.

(The first half of this paper, reprinted on p. 75, throws important light on Dr. Hughlings Jackson's views on speech and its disturbance from lesions of the brain. He deals with emotional and intellectual speech, "verbalizing," internal speech and the power of forming propositions.

The second half contains nothing about speech defects.)

1874.

[25] "On the Nature of the Duality of the Brain," *Med. Press and Circular*, 1874, vol. i, pp. 19, 41 and 63.

(Reprinted on pp. 80, 87, 96.)

[26] "Temporary Affection of Speech (Aphasia), 'Aphasic' Writing," *Brit. Med. Journ.*, 1874, vol. i, p. 574.

(A case of repeated cerebral vascular attacks with aphasia and agraphia. A specimen of handwriting is given.)

[27] "A Case of Right Hemiplegia and Loss of Speech from Local Softening of the Brain," *Brit. Med. Journ.*, 1874, vol. i, p. 804.

(Case of embolic hemiplegia with aphasia in a man, aged 28. An excellent report of what he could and could not say, with a good account of the *post-mortem* appearances.)

1876.

[28] "Case of Cerebral Tumour without Optic Neuritis and with Left Hemiplegia and Imperception," *Roy. Lond. Ophth. Hosp. Reports*, 1876, vol. viii, p. 434.

1878.

[29] "Remarks on Non-protrusion of the Tongue in some Cases of Aphasia," *Lancet*, 1878, vol. i, p. 716.

(Reprinted on p. 104.)

1879.

[30] "On Affections of Speech from Disease of the Brain," *Brain*, 1879, vol. i, p. 304.

(Reprinted on p. 107.)

[31] "On Psychology and the Nervous System," *Med. Press and Circular*, 1879, vol. ii, pp. 189, 239, 283, 409, and 429.

(Here he states his views on psychology, and utters a warning against the fallacious terms in use at the time. One paper is devoted to what he understood by Subjective and Objective, another to Ideation and Perception.)

1880.

[32] "On Aphasia with Left Hemiplegia," *Lancet*, 1880, vol. i, p. 637.

(The case of a left-handed sailor with aphasia and left hemiplegia; he could copy and write to dictation but could scarcely write at all spontaneously.)

[33] "On Affections of Speech from Disease of the Brain," *Brain*, 1880, vol. ii, pp. 203 and 323.

(Reprinted on pp. 130 and 147.)

1884.

[34] "On Evolution and Dissolution of the Nervous System," The Croonian Lectures for 1884, *Lancet*, 1884, vol. i, pp 555, 649 and 739; *Brit. Med. Journ.*, 1884, vol. i, pp. 591, 660 and 703.

(We have not reprinted these lectures because they are not directly concerned with affections of speech. But they enunciate the general laws which govern the activity of the central nervous system, and contain many instances drawn from disorders of speech.)

1887.

[35] "An Address on the Psychology of Joking," *Lancet*, 1887, vol. ii, p. 800.

(*Proceedings of the Medical Society*, 1888, vol. xi, p. 1.)

1893.

[36] "Words and other Symbols of Mentation," *Med. Press and Circular*, 1893, vol. ii, p. 205.

(Reprinted on p. 175.)

[NOVEMBER, 1915.]

BRAIN.

PART III, VOL. 38.

POSTURAL ACTIVITY OF MUSCLE AND NERVE.

BY C. S. SHERRINGTON.

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I.—INTRODUCTORY.

PROMINENT in the volume of *Brain* for 1888 is a discussion on Muscular Hypertonicity. At the opening of that discussion Hughes Bennett furnished a definition of "reflex muscular tonicity" as "that slight constant tension which is characteristic of healthy muscle." Thus simply expressed that is, I think, the current conception which still generally prevails. Simple though it is, it is one which a teacher of physiology finds difficulty in illustrating to students, and the statement remains vague to them. The almost complete absence of numerical measures of tonus, the lack of assignment to it of any clearly purposeful rôle, the want of demonstration of it in the various laboratory exercises on muscle—almost always excised frog's muscle—imbue the phenomenon with a certain mystery unwelcome to those desirous of clear facts for direct use in future practice. To tell the inquirer that tonus serves to obviate the muscle's "taking up slack" when it enters on the performance of a movement, seems rather thin-spun when the class-room exercise has measured the latency of toneless frog muscle as one-hundredth of a second. The further suggestion that the katabolism accompanying the tonus makes it an important means of heat-production—the so-called chemical or thermal tonus—suggests that the contraction tonus is mere by-play in the thermotaxis of the organism; and the explanation comes all the more as a surprise since the chemical turn-over adjunct to the mechanical tonus has been shown i years to be very small.

Renewed inquiry on a larger basis is now lending more pr the phenomenon of tonus. But, as is natural in a phase of t some confusion obtains. That this is so a quotation may show ing recently, Matula [21] says: "By reflex tonus is understoo paper that persistent weak activity of the musculature which by the stimuli continually reaching the body, that is, 'tonus' i geest's old sense. I find it necessary to state this although obvious, for the word 'tonus' is certainly one of the most terms in physiology. Under it each writer understands s different, if indeed he is in a position to supply any sharp at all of the conception."

The confusion seems partly traceable to want of touch inquirers engaged upon the phenomenon in fields of observat ingly rather wide apart. The publications of zoologists and do not commonly meet quickly. An aim of the present article to correlate a number of such observations and tentatively to pi together.

A fairly literal meaning attaching to the term "tonus" is, "mechanical tension." In this sense it fits well the slight stea ing tension so characteristic of muscles in their state of reflex This meaning of the term is evident in the definition quo Hughes Bennett. But in its early use, by J. Müller and writer period, the term carried also or soon came to carry the impli "automatism."

One meets the term in Humboldt's [15] "Researches o and Nerve" of 1797; there it is applied to nerve, and though occasionally, evidently refers to some then current view which an intrinsic activity of the nerves and nervous system generall meaning outlived considerably the discovery that the tonus Müller [27] spoke was reflex and not the outcome of auton nerve-centres. The connotation of the term has drifted directions from the literal meaning of mechanical tension. It to the activity of nerve-centres, thus: "tonic nerve-centres, discharge of nerve-cells" and the "bio-tonus" of living ma these departures there is always more or less prevalent the sig "lasting" and "enduring" muscular or nervous action as c with evanescent or passing. To-day we commonly speak of t of a muscle or nerve as tonic when we wish to indicate that i tively long-lasting. The term has become somewhat vague by reason of the multiplicity of meanings attached to it. But one service expected of a technical term is that it should be precise and unequivocal.

And the conception itself, as well as the term, labours in one important respect under vagueness. Physiology pursues analysis of the reactions of the body considered as physical and chemical events: but, further, it aims at giving reasoned accounts of the acts of an organism in respect of their purpose and use to the organism *qua* organism. This may be called a teleological aim, yet belongs to a teleology not foreign to the scope of natural science. In animal behaviour the more complex the act the less equivocal usually its biological meaning. The physiologist in analysing animal reactions seeks, as a rule, components more elementary than those toward which the "behaviourist" directs his work. Thus, the "behaviourist" examines a train of acts characterizing some instinct; he seeks to describe the sequence of events from their outset in initiatory stimuli onward to movements and turning-points of movements often each one of high complexity and intricate co-ordination, and yet each one sufficiently unitary to serve for his present purpose as one separable piece of the train of behaviour he is fractionating. The physiologist aims at yet simpler units, for instance at the characteristics of a synaptic function, or the dissociation of the periodicity of a nerve-centre's activity from that of a muscle's activity. He takes for his problem reflexes usually much simpler than those studied by the "behaviourist." He may choose some reaction which itself is but a partial factor in a single act a whole train of which form the subject-matter of the "behaviourist." Yet every reflex is in its own measure an integral reaction, and is purposive in that it bears some biological purport for its organism. Every reflex can, therefore, be regarded from the point of view of what may be called its "aim." To glimpse at the aim of a reflex is to gain hints for further experimentation on it. Such a clue to purpose is often difficult to get; and attribution of a wrong meaning may be worse than absence of all clue. But the difficulty is generally inversely as the complexity of the reflex. Thus the larger the muscular field involved in the reflex effect the plainer usually its purpose. A slight movement confined to a single limb or, in appearance, to a single muscle, a transient rise of arterial pressure; these observed alone lie open to many interpretations and admit of no security of inference. They are fractional reactions which may belong to any of many general reactions of varied aim. Thus reflexes observed in paraplegic man have been notoriously difficult to refer to their functional purpose. On the other hand, in lower animals where depression of spinal function and spinal shock are less, the ampler reflex actions, embracing by irradiation wider groups of associated muscles, often write their own meaning clear, and can indeed give a clue to the meaning of the analogous reactions less obviously decipherable in man.

When the spinal dog in response to an irritative stimulus of the scapular skin brings the hind foot to the irritated point and scratches there, the purpose of the reaction is clear. And a reflex of simple kind may have as much purposive completeness as a complex one. Thus, the reflex emptying a viscus, though it involves but a restricted field of musculature, may be as complete for its purpose as is such a reflex as walking, which involves skeletal musculature practically throughout the body.

Further, among data helpful for assigning its purpose to a reflex is the fashion of its elicitation, the nature of the adequate stimulus. That a faradic stimulus applied to the back of the tongue in a decerebrate cat interrupts the rhythm of the respiratory movements, is an observation which may leave us in doubt as to the biological meaning of the result. But the same result, when caused by putting a few drops of water on the back of the tongue, suggests an obvious "purpose," and the interpretation is clinched by the reflex swallow which ensues.

As to muscular tonus much of it is reflex. To know the biological purpose of such a reflex reaction is to have suggestions for lines along which to investigate it. The question arises, does muscular tonus carry the same biological meaning in all its examples; or does it in some cases meet one purpose, in other cases meet another? The decipherment of what biological meanings its various instances possess should help toward obtaining a broader standpoint for evaluating the whole phenomenon itself. A step toward this is to consider manifestations of it in particular cases. In the first place a field of musculature may be taken which is skeletal, because although complex its very specialization reveals the more clearly the particular purpose it effects.

II.—SKELETAL MUSCLE.

By Johannes Müller the term "tonus" was employed to denote a certain steady slight contractile tension which he regarded as the characteristic condition of normal skeletal muscle when not engaged in the performance of any specific act. He thought this slight steady tension the outcome of an influence continually imparted from the nerve-centres of cord or brain. Whether this action of the nerve-centres was of automatic or reflex production he left open. Marshall Hall [14] considered the spinal influence maintaining the steady closure of openings by sphincters to be a reflex tonus, though, it is true, he gave no further account of its source or of the stimulus exciting it.

The early experiments directed toward demonstrating the state of tonus of skeletal muscle met with divergent results. Their plan of observation was to look for a slight elongation of the muscle (frog) after severance of the muscle's nerve. Heidenhain, Auerbach, Schwalbe and Pflüger were among those who failed to get clear evidence of any such lengthening. In the fifties of the last century the existence of "tonus" of skeletal muscle seems to have become doubted by many physiologists. But in 1860 it was reaffirmed by the observations of Brondgeest [7], of Utrecht. Brondgeest showed that in the frog if the afferent spinal roots of one hind limb be severed, the corresponding limb when the frog is held vertical is less flexed than is the fellow sound limb. He argued that the greater flexion of the sound limb is due to a reflex tonus of its muscles. The observation was soon recognized as trustworthy. The failure of previous observers to find any definite elongation of the skeletal muscle on nerve severance remained, however, unaccounted for. Twenty years later a critical summary of the position by Eckhard [11] concluded that "tonus" of skeletal muscle had been demonstrated for but few muscles, and was far from having been shown present in all muscles and at all times. As to the source of reflex tonus in those muscles in which it was present Eckhard inclined to accept the suggestion that the source is cutaneous, the skin at one point or another being continually open to numbers of stimuli, which, although not individually sufficient to cause reflex movement, yet by their summation keep up a more or less continuous additive mild reflex effect. And the skin as a source of the reflex tonus of skeletal muscle is still apparently occasionally accepted.[1] But Mommsen [24], in 1885, pointed out that the Brondgeest phenomenon persists after the limb has been stripped of its skin.

The tonus of skeletal muscle can be studied favourably in the mammalian preparation. Removal of the brain from the posterior colliculi forwards in the cat provides an excellent tonic preparation of the extensor muscle of the knee. This tonus is still retained by the muscle to the full after severance of all the skin nerves of both hind limbs. Further, the muscle still retains its full tonus after severance of *all* nerves of both limbs excepting only the nerve of the tonic muscle itself. That nerve consists, of course, of fibres afferent as well as efferent. The afferent fibres are traceable partly from the muscle's tendon, but mainly from the muscle itself. These afferent fibres reach the spinal cord *via* the dorsal (posterior) roots of two spinal nerves (the fifth and sixth lumbar in the cat). If these two afferent dorsal roots are severed, the tonus at once vanishes from the muscle, although the corresponding ventral roots containing the motor fibres for the muscle remain intact, and although all the other nerves of the limbs remain intact as well. And similar experiments with other muscles exhibiting tonus, e.g., gastrocnemius, semimembranosus, triceps, supraspinatus, meet the same result. In each case the tonus of these muscles requires the afferent fibres of the tonic muscle itself, and in the decerebrate cat preparation no other afferent fibres than those of the tonic muscle itself are actually essential for the exhibition of the tonus.

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[1] W. Fröhlich, *Zeitschr. f. Allg. Biologie*, 1911, ix, 1, 63.

The tonus of these muscles in this decerebrate preparation is not a phenomenon requiring for its detection and demonstration any refined apparatus, or indeed any apparatus at all. It and its features are palpable and obvious; graphic records of it are obtainable by relatively coarse methods. The extensor muscle of the knee, vasto-crureus, lends itself well to the purpose. That and the three other muscles mentioned above are specimens of the tonic muscles in this preparation. But many other muscles in this preparation show the tonicity as well. In the hind limb the distribution of the tonicity in the musculature shows the following feature. If the reflex act of stepping is examined as it may be both in the spinal and in the decerebrate preparation (cat), the act is found to consist of two phases; in one phase—the flexion phase—the foot is lifted slightly from the ground, and the limb is swept forward by flexion of hip, accompanied by flexion at knee and ankle, so that the foot may clear the ground in its advance. In this phase all the flexor muscles of the limb are excited to contract, and all its extensor muscles are inhibited by reflex inhibition. The other phase—the extension phase—is that in which the foot being in contact with the ground, the limb is straightened at knee and ankle, and kept from bending under the body's weight by the extensors of those joints; and at the hip, the extensors using the foot's *point d'appui* against the ground as a fulcrum for the leverage by the limb, push the body forward. In this phase all the extensors of the limb are in active contraction, and the flexors are reflexly inhibited. The distribution of the reflex tonus of the decerebrate preparation in the musculature of the limb is exactly to those muscles which are in active contraction in the extensor phase of the step, to those and to no others.

In the forelimb, though this analysis of the exact distribution in the musculature of the extensor phase of the step has not been so completely made, the analysis, so far as it goes, shows again exact correspondence between the musculature exhibiting the tonus and those engaged in contracting in the extensor phase of the step. The two phenomena involve, and are confined to the same group of muscles. It is in the extensor phase of the step that the limb is supporting the weight of the body.

The distribution of the tonus in the limb musculature reveals, therefore, arrangement on a plan of strict co-ordination. It is, however, not confined to the musculature of the limbs. It is as markedly present in various other regions. In the trunk it obtains in the muscles which bend the vertebral column upwards (opisthotonos); in the neck in those muscles which lift (retract) the neck and head; in the caudal region in those which lift the tail; in the head in those which close the jaw. It is not present in those muscles which bend the spine downwards, droop the neck and tail, flex the head, depress the jaw. It seems to be present, but of this I am not entirely sure, in the ventral muscles of the abdominal wall. Evidently, therefore, the distribution of this reflex tonicity embraces just those muscles whose contraction tends in the erect position of the animal to counteract the effect of gravity on the various several regions, the muscles which prevent those parts, and the animal as a whole, from sinking to the ground. And from the muscles antagonistic to these the reflex tonicity is absent. In other words, the reflex tonus obtains in, and is confined to, those muscles which maintain the animal in an erect attitude. That this is so may be demonstrated by setting the decerebrate preparation on its feet; it is then seen that the preparation stands. Thus this reflex tonicity, which when seen in a single isolated muscle prepared for the myograph, does not carry on the face of it any very obvious biological purpose, does carry a clear and unmistakeable biological purpose when the phenomenon is followed in the musculature as a whole. The reflex tonus is postural contraction. Decerebrate rigidity is simply reflex standing. The reflex tonicity of the skeletal muscles of the decerebrate cat and dog is shown by its co-ordination, its effects, and its distribution in the musculature, to be a reflex which differs from the reflexes more commonly studied mainly in this, that the latter execute *movements* while this maintains *posture*. The reflex tonus is in short reflex posture, and in this case the posture maintained is that of *standing*. And the reflex posture is modifiable on the supervention of certain additional stimuli, and the modifications of posture thus obtained are so intelligible as forms of standing adapted to particular purposes that they carry on the face of them that significance. If the head of the reflexly standing decerebrate preparation be forcibly flexed, the postural contraction of the extensor muscles of the fore limbs is inhibited, and the animal's fore quarters sink, while at the same time the postural contraction of the extensors of the hind limbs increases, raising the hind quarters. The preparation thus assumes the attitude of a cat looking under a shelf. On the contrary, if the head of the preparation is passively tilted up and back the postural contraction of the extensor muscles of the fore limbs increases, raising the fore quarters, and at the same time the postural contraction of the extensors of the hind limbs is diminished so that the hind quarters sink. The preparation thus assumes the posture of a cat looking up to a shelf. There goes further with each main posture of the head even passively imposed upon the decerebrate preparation a corresponding reflex modification of the reflex posture of the limbs. Magnus and de Kleijn [20], to whom is owing the elucidation of this subject, have described these fully, and shown their constancy, and shown further that the centripetal impulses causing these reflex modifications of the reflex standing are traceable for their one part to the otic labyrinth, for their other part to the deep afferent nerves (proprioceptors) of the neck itself. These experimenters have succeeded in separating the reflex results from the two sources, and have thus determined what part each source plays in the combined effects which under natural conditions are those of usual occurrence.

As mentioned above, the afferent nerves producing and maintaining this postural reflex of standing are the afferent nerves of the posturally contracting muscles themselves. The whole reflex posture of standing is thus one great compound reflex built up of a number of component reflexes. To the making of the total reflex posture there goes the postural reflex of each limb, similarly the postural reflex of the neck, of the trunk, of the tail, of the head. And the reflex posture of each component region is in large measure separable from that of the rest, and is capable, within limits, of modification, although remaining still contributory to the general posture of standing. This is in accord with the natural occurrence of, for instance, such a modification of the erect posture as "sitting" in the cat or rabbit; a posture half-way between lying down and standing, the fore limbs "standing" and the hind limbs lying down.

And the local reflex posture, say of a limb, can be modified by local influences. It is in the examination of these that we meet with exemplification of characteristics of postural contraction to which attention will be drawn in dealing with the postural contraction of muscular walls of the hollow viscera and blood-vessels. In the posturally acting skeletal muscles these characters appear as what have been termed [36] the "lengthening" and "shortening reactions." The "lengthening reaction" and the "shortening reaction" are given by the skeletal muscles of the cat in postural contraction and they are given also by the smooth muscle of the viscera, and of invertebrata. Just as the postural configuration of the knee or elbow is adjustable by these means so likewise is that of the bladder or the stomach.

The "lengthening reaction" of a skeletal muscle engaged in postural action is easily seen in the knee-extensor of the decerebrate preparation and can be suitably examined there. The muscle is isolated by paralysing, by appropriate nerve-section, all the other muscles of both hind limbs. As mentioned above, the muscle (vasto-crureus) when this has been done still maintains its own postural reflex action unabated. For the purposes of the observation the preparation is preferably placed on its side, on the left side if the vasto-crureus chosen be the right. The muscle still retains uninjured its attachment to the patella and, by patellar tendon, to the tibia. Starting with a knee-posture of nearly full extension the tension of the muscle as it holds the knee in this posture is examined by finding what weight pulling the tibia backward—i.e., tending to flex the knee—the postural action of the muscle just counteracts. The observer then with his hand forcibly bends the knee, overcoming the contraction of the extensor; he brings it into a new position, let us say, of nearly full flexion, performing the movement steadily and not too quickly—e.g., so that it occupies a couple of seconds. On then releasing the knee there is usually a slight brief partial movement of it in the direction of return toward extension, but apart from this, which does not alway occur, the limb remains in the new [illegible] the position which was passively imposed upon it. The te[illegible]f the extensor muscle as examined by the weight, is in the new posture practically just what it was before, neither more tension nor less tension than before. But in the new position the length of the muscle is of course greater than it was in the former. In short, the posturally acting muscle has, in result of the extension imposed upon it, assumed a new postural length, and has assumed a greater postural length without exhibiting in its new state of greater elongation any practical departure from its previous degree of tension.

In the same preparation, conversely the shortening reaction may be elicited. For this, starting with nearly full flexion as the reflex posture of the knee, such a flexed posture for instance as is left at the end of a "lengthening reaction" like that just described, the tension of the muscle is estimated by the amount of load-traction it counterbalances on the tibia. The observer then passively extends the knee so as to bring it to, for instance, nearly full extension. On then releasing the limb it is found, although the weight is still exerting practically the same pull toward flexion as before, to remain in the new posture of extension passively imposed upon it. The posturally acting muscle has taken up a new postural length, and is now shorter than before, but exhibits practically the same tension as at its previous length. On examining the tension by the weight it just counteracts, there is found to be practically no alteration of the tension. The skeletal musculature by reason of these lengthening and shortening reactions allows that latitude of pose which is so useful and familiar a feature in natural attitudes. The animal may stand with right foot in advance of left, or left in advance of right, or with the two feet abreast of each other; all these differences in detail from local posture to local posture are compatible with the general posture of standing in the animal as a whole. The postural reflex contraction is plastic in this sense, hence the term *plastic tonus* [36] has been applied to it. The skeletal muscle in this form of reflex contraction can quite readily adjust itself to different lengths while counteracting one and the same load.

In the above examples of the lengthening and shortening reactions of skeletal muscle, the alterations in posture, to which the limb was subjected in showing its power of adjustment, were alterations imposed by passive movement. The observer moved the limb into the new attitude which the reflex postural contraction then took up and lightly fixed. But the reflex adaptation to the new posture occurs just as well, or better, when the changed position of the joint is brought about by active reflex movement excited, fo[illegible]e, by faradizing an afferent nerve-trunk. Thus in the postu[illegible]preparation when a reflex extension movement of the knee has been provoked, the shortening reaction appends itself to the reflex contraction, and on discontinuing the stimulus which caused the extension movement the extensor muscle still remains shortened, and the knee still continues in the extended position. This lends to reflexes of the decerebrate preparation a character recalling some features of catalepsy. The limb brought by active or passive movement into a new posture when released remains in that new posture.

Again, the animal being on its side and the postural contraction of vasto-crureus just counteracting a certain weight tending to flex the knee by a cord running over a pulley and attached to the tibia, let an afferent nerve, appropriate for reflexly inhibiting the postural contraction of the muscle, be stimulated with a series of mild break shocks at equable intervals. Let the intensity of the stimulation chosen be such that each shock causes a minimal brief inhibitory lengthening of the posturally contracted muscle. The muscle elongates in a gradual minutely step-like manner. Throughout the steps of the lengthening process, and at the end of that process, when the electrical stimulation is withdrawn and a considerable total lengthening of the muscle has taken place, the postural contraction still just counteracts the same weight as it had just counteracted initially at the outset of the observation. The "lengthening reaction" obtains, therefore, with reflex inhibitory lengthening of the muscle as well as under forcible passive lengthening of the muscle.

That the *tension* of the posturally acting
approximately unaltered over a lar
muscle in different postures of the
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the tibia, 11 cm. from the kne
angle of 110°, and whe
co n just cou

load in the different attitudes of knee. The hardness of the muscle as actually measured was practically the same in all the varied lengths of muscle under the different poses of the joint.

The observations on the tonus of skeletal muscle in the mammal, therefore, go to show that the phenomenon is in skeletal muscle nothing more nor less than postural contraction. Why did the search for tonus in the skeletal muscles of the frog meet with such discrepant results and equivocal interpretation? In that search it seems to have been regarded as unimportant what particular muscle was selected for the search, the supposition being implicit that tonus would attach either to all muscles or to none. But if we regard tonus as a contraction of muscles engaged in the execution of a definite co-ordinate reflex, a reflex differing from reflexes ordinarily examined only in its function being posture and not movement, we shall expect reciprocal innervation of antagonistic muscles to obtain, and in that case if one set of muscles are in reflex contraction their opponents will not be in contraction but under reflex inhibition. That in postural reflexes reciprocal innervation of antagonists does hold has been found in fact to be the case. Thus in reflex standing the opponents of the posturally contracting anti-gravity muscles, the flexors as they may in brief be termed, exhibit no postural contraction, and the stronger the reflex posture the less trace of contraction may these latter be expected to show. It is noteworthy that in Brondgeest's [7] experiment demonstrating reflex tonus
hind limbs of the frog, the muscles which exhibit the tonus are
reverse of what obtains in the decerebrate cat, dog,
al posture of the frog is squatting,
head is somewhat lifted, the fore
eight of the forepart of the animal,
ly full flexion, doubtless in appro-
spinal frog retains as its reflex
was long ago noted by Volkmann
part of the spinal cord in
peration h ded

specially studied by v. Uexküll [39] and J. Parnas [30], the movement muscle executes also the rhythmic adduction of the shell-valves in swimming. Pecten being a monomyarian has in appearance only a single adductor muscle; but examination shows this to consist of two parts, one the glassy "movement muscle" the other the semi-opaque "postural muscle." How completely the closed posture of the shell depends upon the latter is demonstrated by severing the visceral nerves supplying the postural muscle. The "movement" adductor muscle remains still unparalysed. The shell then stands open, and to various stimuli, which normally cause the shell to shut and remain shut, the shell responds more readily than usual by closure, but the closed posture is not maintained, and the shell gapes again at once.

With the intact Pecten, if a penholder, put between the open lips of the shell, touches the parts within, the shell-valves close upon the intruder and hold it tight. The wooden holder is so tightly gripped that it can be extracted only with difficulty, but that done the shell-valves remain at the same degree of partial closure, they do not close further. Yet any attempt to pull them farther apart reveals that to that they oppose a very great resistance. The movement of adduction was executed by the contraction of the "movement adductors": their short-lasting contraction then ceased, but the degree of adduction of the shell-valves thus arrived at is then maintained as a posture by the postural contraction of the postural muscle. The speed with which the postural muscle can by contraction shorten its length is relatively slow as compared with that of the movement muscle. But the postural muscle exhibits the phenomenon already adverted to in the extensor muscles of the limbs and neck in postural contraction, namely, the "shortening reaction," seen also in sipunculus and in the contractile skin of holothuria. If the shell-valves of Pecten be passively partly closed or fully closed they remain so. Their postural adductor takes up and retains the closed shell posture passively imposed upon it. It does so also when the shell is actively adducted by the movement adductor. V. Uexküll discovered a means of exciting a reflex opening of the shell. Experimenting upon this, he found that lengthening of the check-adductor in its postural action can be brought about reflexly.

The mechanical elasticity of the postural adductor is very small. Suppose a partially open Pecten after severance of the movement adductor and loaded with a weight so attached as to tend to pull it open farther, though failing to do so. On cutting the string attaching the weight, the shells do not perceptibly spring nearer together. Nor is there any proportion between the power to move the weight and the power to support the weight. The postu[illegible]ductor can support for hours a weight it cannot raise. A weakly [illegible] which in the aquarium does not and cannot close its shell is, [illegible] shells be closed by the observer and kept in the closed posture for a few minutes, found able then to keep the shells in that closed posture against the pull of the elastic opening ligament.

The existence in various invertebrata of muscles separately differentiated for execution of movements and for maintenance of posture respectively seems without parallel in the skeletal musculature of vertebrates. In the latter, one and the same muscle is used for the two purposes, though some muscles are predominantly concerned with the one, some with the other function. Perhaps the nearest approach to muscles of purely postural function in mammals are the sphincter muscles controlling orifices. But in most of the more complex reflex acts the reflex while employing some muscles for execution of movement simultaneously employ others for maintaining posture. Thus, in the scratch reflex while one hind limb is engaged in the rhythmic scratching movement, the reflex employs the muscles of the other three limbs and of the neck and head for the maintenance of a characteristic posture which continues so long as the reflex scratching continues. Similarly, in a powerful nociceptive flexion reflex, while the stimulated limb maintains the attitude of flexion the other limbs frequently perform stepping movements, just as in the intact animal that has stepped on a thorn the injured foot is held folded up and the other legs run away. And with these skeletal muscles one and the same muscle may at the same time exhibit both postural contraction and phasic or movement contractions. Thus, in the scratch reflex, there is required in order that the hind limb reach the neck and apply there its scratching movement a certain posturing of the limb as well as rhythmic movement of it. For this there is demanded some postural flexion of the hip. It is found that the sartorius muscle, which is a flexor of hip and knee, shows in this case a well-marked degree of steady postural contraction as well as, over and above that, the characteristic four per second rhythmic contraction of the scratching movement itself.

A question which arises is whether in all cases the reflex postural action of skeletal muscle depends normally upon the afferent nerve of the posturing muscle itself. In the reflex posture of standing exhibited by the decerebrate cat that does largely seem to be the case. Severance of the afferent spinal roots of both hind limbs in the dog (Bickel [2]), renders standing with those limbs impossible for a considerable period; but in the course of time the animal becomes able to support itself upon them, a compensation traceable to labyrinths, cerebellum and motor region of cerebral cortex. In the pigeon, severance of the afferent roots of both hind limbs makes standing impossible (Trendelenburg). Severance of the afferent roots of one leg impairs also the flexion posture assumed by the hind limb during flight. Section of the afferent roots of the wing affects little, if at all, the folded posture maintained by the wing when not in flight—e.g., during standing or walking. Nor has the source of the postural contraction of the flexors of the wing been found. Here, as in the case of the iris, the postural contraction if, as is presumable, reflex, lies in receptors not those of the contracting muscles themselves, is, in short, allogenous not autogenous.

That receptors other than those of the contracting muscles themselves can be adjuvant to the reflex postural action maintained by these latter is evidenced in many observations. For instance, Ewald [12] has shown that the postural closure of the pigeon's beak is impaired by destruction of the labyrinth; and after splitting the lower bill into its two lateral halves he found that destruction of the right labyrinth weakened the postural contraction of the right half much more than that of the left: the movement of closure was often fully executed, but with the right half the closed posture was less powerfully maintained; this was clearly demonstrable by hanging upon each of the separate halves one of a pair of equal weights—the right half did not maintain the closed posture under so heavy a load as did the left: an observation recalling those above mentioned on the pecten shell.

And Magnus and de Kleijn [20] have shown the existence of a number of important postural reflexes of labyrinthine origin affecting the extensor muscles of the limbs. To excite the labyrinth they employed modes of stimulation natural to it—"adequate" stimuli—namely posings of the head in regard to the direction of gravitational force. The labyrinth is a receptive organ specialized for reacting to gravitation force, hence it initiates sensations reporting on the spatial relation of the head to axes running through it and the earth's centre, in short, to the vertical, and the reflexes which the labyrinth initiates consist in adjustments of the head, including eyeballs and jaw, to the vertical. The action of the muscles in the posture of standing is anti-gravitational. Hence it is not surprising that the labyrinth reactions should be related to and influence the anti-gravitational reflex of standing, though this latter has its origin in the receptive organs of the standing and neck increases the extension of the fore limbs and lowers the hind quarters just as when the normal animal looks up at a high shelf. The bending of the neck and head to the right causes increased extension and advancement of the fore limb as normally occurs when the animal modifies its attitude for gazing round to the right. The increase of extension of the limbs when the preparation is inverted is also what happens in the normal animal when it is inverted; of course, the normal animal usually struggles against such an attitude being forced upon it, but, when that resistance is over, it, just as Verworn has shown in the guinea-pig, keeps the limbs extended strongly upwards, and may, by what is sometimes termed hypnosis, maintain that protective posture for a long time, even when left to itself and free from restraint.

That in the reflex standing of the decerebrate cat and dog, the postural contraction of the anti-gravity muscles which is its essential mechanism, is in the main a proprioceptive reaction whose afferent nerves are those of the anti-gravity muscles themselves is clear. As to the nervous centres involved, the following seem the main facts: In the dog after transection of the spinal cord in the hinder thoracic region, when in the course of some months, the depression of spinal function, termed spinal shock, has subsided the hind limbs are not rarely able to stand. They can bear erect the superincumbent hind-quarters, so much so that the observer placing his hand under the hind feet can find that, on lifting the hand suddenly the erect posture of the hind limbs is sufficiently strong to maintain itself, while the hinder portion of the animal is danced up and down by the hand so placed. And the spinal hind limbs will maintain their standing posture for half an hour at a time. But it is subject to sudden lapses the cause of which may not be obvious, although it is often some evident stimulus to the foot or elsewhere exciting intercurrent reflex flexion of the limb. But although spinal centres isolated from prespinal are, after the period of spinal shock has passed, able *per se* to maintain the posture at least in fair degree in cat and dog, they cannot in the period immediately following the isolating transection. Normally, some prespinal centre, or probably several centres, is adjuvant either in the sense that the postural reflex besides employing the spinal centres employs prespinal as well, or in the sense that the spinal centres which the reflex employs are kept up to the mark for that reaction by influence exerted on them by prespinal. As to where these prespinal centres lie the following can be said. Unessential for crude maintenance of the act of standing in cat and dog are the whole fore-brain and mid-brain back to and inclusive of the posterior colliculi. Likewise the labyrinths are not necessary since they can be destroyed and the reflex posture persist. Further, the cerebellum can be removed without the posture being annulled. Evidently the prespinal centres necessary lie in the pontine region or bulb, or both, though mainly in the former.

But though the neural mechanism of the standing posture does not essentially demand for its crude performance those mid- and hind-brain regions, these do afford it assistance, and as Magnus and de Kleijn's observations prove for the octavus nerves provide for it reflex adjustment in manifold important ways. And Weed's [40] experiments indicate that the cerebellum commonly lends it a large amount of support and doubtless of refined adjustment and correlation too.

A further question is whether in *all* cases the reflex tonus or postural action of striped muscle has its source in the afferents of the posturally acting muscle itself. A departure from this rule might appear to be given by the extrinsic muscles of the eyeball. These are pre-eminently postural in their function, since the gaze is in most cases typically a posture. The third, fourth, and sixth cranial nerves are usually regarded as being purely motor nerves. But it has been somewhat recently shown that a large number of the fibres of these nerves are afferent and supply receptive nerve-endings to the recti and obliqui muscles and their tendons. The common occurrence of squint in tabes is also evidence that the postural action (tonus) of these eyeball muscles, is based on reflex action subserved by the afferents of these muscles themselves. The squint would be evidence of a hypotonia analogous to the hypotonia of tabetic limbs.

There is, however, a case in which the reflex postural action of a muscle, not a striated one, is dependent in large measure on afferents distributed not to itself. The sphincter pupillæ, like other sphincters, exhibits marked postural action, and its degree of contraction, its tonic posture, has its reflex origin largely in the retina. Here the reflex arc, as is well known, involves certain of the fibres of the optic nerve, and central stations lying in the anterior colliculus. Here the natural stimulus exciting the reflex posture is light, and the posture itself has, of course, as its *raison d'être*, the adjustment of the organ of which it is a part in regard to that very agent which is its source of stimulation. If we apply that inference to the case of the postural action of the limb muscles in, for instance, standing, the *raison d'être* of their postural action is the adjustment of the length of the muscle against a stimulus which is gravity acting in the form of mechanical tension upon the muscles themselves. In the case, therefore, of the limb muscles and their reflex postural action in standing, we should expect the afferents of the muscles themselves to be the media of the reflex and the agents stimulating their end-organs to be mechanical strain. That brings the difficult question, what is the natural stimulus which excites the reflex postural action of the muscles in such a case as standing?

Evidently it is some state or process resident in the posturing muscles themselves. The afferent nerve-fibres gathered up in the afferent nerve of the muscle are traceable peripherally to nerve-endings, some of which are distributed to the muscle proper, others to its tendinous, aponeurotic, and fascial appurtenances. And there is evidence that the afferent nerve of a muscle contains admixed in it constituent fibres of various reflex effect. The afferent nerve-fibres of an extensor or anti-gravity muscle appear to be of two functional kinds. One kind, a, produces reflex inhibition of its own muscle and of other allied extensor muscles and reflex contraction of flexor muscles antagonistic to the muscles it inhibits. This kind usually dominates in the reflex reaction excited from the afferent nerve by faradic stimulation of moderate or considerable intensity. The other kind, β, produces reflex contraction of its own muscle, an extensor muscle; and this kind is excited by very weak or brief faradic stimulation, and even with moderately strong faradic stimuli often makes its effect seen at the onset of the reflex response. A form of electric stimulation which seems to suit this β kind better than does faradic stimulation is serial brief non-abrupt galvanic currents such as are provided by the rotating rheonome. Evidently the a kind, if it cannot excite contraction of its muscle, cannot be answerable for the knee-jerk which there is now good ground for regarding as a true and, of course, proprioceptive reflex. To the β kind, therefore, the knee-jerk can be relegated. If it is the β kind which is responsible also for the production of the postural contraction of the muscle, and the special amenity to electric stimuli of mild intensity supports this probability, that would explain the association of the knee-jerk with tonus or, as it is preferred to say here, postural contraction. The knee-jerk though not a tonic or postural reaction itself,[1] is in many circumstances a significant test for the tonicity arc, that is, for the arc on which depends the reflex postural contraction of the muscle.

A feature of the knee-jerk related to postural reflexes, very the muscle by stretching it through one of its mobile attachments; and also on elongation of the muscle under relaxation by reflex inhibition. By the former method there is increased mechanical tension of the muscle, by the latter there is no increase of its mechanical tension. But the fleshy part of the muscle undergoes a similar change of form in both procedures, that is, the muscle-fibres become longer and narrower under both procedures. This would suggest that the afferent nerve-fibres concerned in the lengthening reaction have their receptor organs in the fleshy part of the muscle and not in the tendinous. The receptive organs belonging to the fleshy part of the muscle are in the main the spindles; the Golgi organs belong to the tendon.

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[1] See Sternberg, M., "Die Sehnenreflexe," 1893. Vienna.

As regards the shortening reaction, it, like the lengthening reaction, can be obtained in either of two ways—by passively approximating the points of attachment of the muscle, or by causing the muscle to shorten itself actively by reflex contraction. By the former method the muscle in the progress of its change to a shorter length undergoes no increase of tension, by the latter method it does. But the shortening reaction ensues by both methods. The tendinous portion of the muscle, although fully participating in tension changes of the muscle, participates relatively little in the form changes, these latter affecting chiefly the fleshy portion. This suggests that the afferent nerve-fibres concerned with the shortening reaction are, as in the case of the "lengthening reaction," those which have receptor organs in the muscle tissue proper; the spindles and not those connected with the tendon organs.

The abdomen is a muscular walled chamber. Kelling [16] examined the intra-abdominal pressure in the dog before and after the animal's taking of a copious meal. He found the intra-abdominal pressure altered very little after the intake of a volume of food amounting to 50 per cent. of the total contents of the abdomen in the fasting condition. Thus, after twenty-four hours' fast the water manometer showed a pressure of 2 cm. The dog then ate 300 c.c. of meat. The dog's girth at epigastrium measured then 40 cm. as against 35 cm. previously, and at penis 30 cm. as against 27 cm. On the introduction further of 100 c.c. of air into the abdomen the manometer registered 4 cm. pressure. On then killing the animal the total abdominal contents, viscera and their contents, amounted to 950 c.c.; the stomach was found to contain 380 c.c. of food. Kelling infers from his observations that the additional volume of contents must be made room for by reflex expansion of the abdominal wall—i.e., by reflex adjustment of the postural contraction of the abdominal muscles. The diaphragm being one of the muscles limiting the abdominal chamber, it may be recalled that Dittler found that the action-currents of the diaphragm did not subside even at the end of its expiratory period; that is, a residual degree of contraction still persisted in it; this suggests a postural contraction which would contribute toward the regulation of the capacity of the abdominal chamber.

The evidence obtained from the skeletal muscles shows, therefore, that when operated reflexly they have the power of exhibiting the same tension for different degrees of active length, for they are able to counterbalance just the same extending force whatever, within a wide range, their contraction length at any given time may happen to be. We are so accustomed to regard the muscle-fibre as an elastic string that this property of exhibiting different lengths, while exhibiting one and the same degree of tension, appears contrary to our fundamental notions of muscular activity. It is a property of which the skeletal muscle, when not under the action of nerve-centres, shows no trace, and it is a property which, so far as has yet been found, is little or not at all exhibited by muscle under artificial stimulation of its motor nerve. It is, however, a property which is especially present in skeletal muscle when the nerve-centres are operating it for the maintenance of posture. It characterizes the postural contraction of skeletal muscle, and it does so in the invertebrata as well as the vertebrata. We may next inquire whether other muscles than the skeletal exhibit it; whether the visceral and vascular systems of muscle also give evidence of its occurrence in their activities.

III.—VISCERAL MUSCLE.

Bladder.—In 1882, Mosso and Pellacani [26] published observations upon the urinary bladder, in man and in the dog, difficult to harmonize with the view, then general, which regarded the viscus as simply an elastic reservoir, capable of emptying itself by muscular contraction when under accumulation of its contents it became "full." As to the manner of its evacuation their observations brought little new, but as to its being simply an elastic reservoir their observations were revolutionary. If the bladder be a flexible elastic membrane such as is a rubber bag, it must, under distension by its gathering contents, develop steadily an increasing pressure as the volume of fluid increases within it. Starting from emptiness such a bag will first unfold its flexures, and after that a rise of pressure within it must accompany the stretching of the elastic wall. Leaving aside the hydrostatic pressure of its contents as for our purpose negligible, the bag's pressure will until its flexures are unfolded be practically zero. The zero pressure obtaining at the beginning extends therefore over a short range of capacities the upper limit of which is determined by the size to which the bag can be unfolded without being otherwise extended. For every quantity beyond this the pressure will be positive, and each increase of contents will heighten the pressure though, as experiment shows, not simply proportionally with the increased volume of the bag. The actual amount of increase of pressure for a given increase of contents will depend on the elasticity of the bag-wall.

The relation between increments of volume and of pressure depends not only on the co-efficient of elasticity of the membrane composing the bag, but on certain other factors as well. Osborne [29] has dealt with this theoretically and by experimental observation. Where, as with rubber, the extensibility of the elastic material is great, its dimensions, including its thickness, alter much under the stretch imposed. This complicates the problem of determining the co-efficient of elasticity. It was to lessen this difficulty that Fick, for whom muscular contraction was in the main a problem of elasticity, devised the isometric registration of the contraction of frog's muscle, which allows scarcely any alteration of the length of the muscle even when its tension alters greatly. In thin-walled elastic bags and hollow viscera this change of configuration of the elastic structure is large and creates serious complication for the analysis of results. A further complicating factor is that a subspherical bag may change in general figure as its size alters. And further factors still are certain obscure changes in the physical consistence of the rubber membrane as inflation and deflation proceed. An "initial rigidity" (Osborne) makes the membrane less extensible at first than later. And there is with rubber an elasticity after-action negligible with ordinary solids, which makes the rise of pressure established by an increment of distension tend to fall off and be imperfectly maintained. A time factor, therefore, enters to some extent into the relation between the bag's capacity and pressure. Further, in the bag when distended beyond a certain point the pressure instead of rising further on further distension may actually fall somewhat, though on then lessening the contents-volume there is no re-ascent of pressure. This Osborne inclines to attribute to "initial rigidity." With extreme stages of distension, when the bag is near rupturing, altered relations finally set in, but these, as wanting all likeness to conditions in the viscus, can here be left aside.

Osborne [29] compared with the behaviour of rubber bags that o the urinary bladder examined twenty-four hours after death. In this latter, from its zero-pressure capacity upwards, increments of content produced somewhat rapidly increasing increments of pressure. No "initial rigidity" was found, and no point where further increase of volume was accompanied by fall in pressure. When the distension was pushed to the limit of elasticity of the wall, the gradient of ascending pressure became very steep prior to the bursting point and did not progress as a linear function of the volume. The bladder twenty-four hours after death must, we may suppose, be regarded as dead in so far as concerns the nervous and muscular constituents of its wall unless very special precautions be taken. Such a supposition is borne out by Kelling's observation on the loss of excitability of the muscular coat of the stomach within fewer hours after death. Experiments on the dead bladder would demonstrate not so much the relations obtaining between volume and pressure in the living viscus as in that viscus with a non-operative muscular coat and nerve elements. Thus they furnish a useful control for observations such as those of Mosso and Pellacani dealing, as do the latter, with the mechanical properties of the muscular wall in life.

Mosso and Pellacani [26] found in the living bladder of man and dog little evidence of a zero-pressure even in the "empty" organ, none of a zero-pressure regularly obtaining whenever the capacity was below a certain amount. In a small dog a water reservoir giving a pressure head of 5 cm. water when connected by catheter with the "emptied" bladder *in situ*, usually did not transfer any of its water to the bladder whether the latter were full or empty. The bladder was observed even at rapid intervals to hold very different volumes of water under one and the same pressure. It would exert a pressure of 10 cm. water whether its contents were 10 c.c. or 90 c.c., or a pressure of 15 cm. whether it contained 50 c.c. or 150 c.c. They concluded that in man an intravesical pressure of 18 cm. water is usually accompanied by desire to empty the viscus; and they point out that the stimulus exciting desire to micturate is closely related with intravesical pressure but not closely with the quantity of bladder content—i.e., bladder volume. They found the sensation of fulness of the bladder arise in the same person with various volumes of bladder-content according to circumstances. If, by injection of water into the bladder under a pressure of 18 to 20 cm. water, the desire to micturate were aroused, and if then the evacuation were prevented by closing the catheter for a short time, the feeling of fulness passed off, and on re-opening the catheter the intravesical pressure was found to have subsided below the pressure at which sensation of fulness arose. The same animal's bladder might at a given time contain a larger volume of content at a lower pressure than that at which at another time it contained a smaller quantity, thus, at one time 95 c.c. at 10 cm. pressure, at another 50 c.c. at 15 cm. pressure. Speedy introduction of additional fluid into the bladder caused a temporary rise of pressure which then partly fell off, the viscus settling down to its increased contents under a pressure little above that which it had previously exerted on a much smaller quantity. In short, the viscus responded to the increment of content by a sort of active diastole proportioned to the volume of the increment. Conversely, on withdrawing from the viscus some of its content Mosso and Pellacani observed a passing fall of pressure followed usually by re-establishment of nearly the same pressure as before. The contractile bladder wall followed up the partial evacuation and then held the reduced amount with about the same light tension of grasp as it had previously exerted on the larger.

The authors remarked these unexpected features of the bladder tonus. In terms of tonus their observations were described, and would still be described, somewhat as follows. When the bladder dilated so as to hold more content its tonus was spoken of as lessened. Adhering to the literal sense tonus, however, means tension, and the salient feature of the above reaction is that the vesical tension remains little altered, and what alteration there is lies in the direction of increase. Conversely, when, after some of the viscus content has been withdrawn, the viscus wall has followed the diminution and envelops the reduced content with nearly the same, though usually a somewhat less, pressure than before, what shall be said of the change in tonus? Shall it be called more or less? Mosso, years later on a visit to England, happened to see an experiment with an isolated viscus showing change of tonus. He turned to those of us near with the exclamation, "Ah, tonus, what is tonus? Who can say what tonus is?" Either we have to get rid of the connotation "tension" from our idea of tonus, or else, as making for clearness, to avoid using the term in regard to such reactions of the viscera.

The living bladder, unlike the dead bladder or rubber bag, enfolds its contents with the same light grip whether those contents be ample or little. Similarly the hand when grasping a ball may exert a similar light clasp, whether the size of the ball be larger or smaller. The bladder sometimes enfolds a large volume with a lighter pressure than it may exert at another time on a smaller volume. In speaking of the hand clasping a handful we do not employ the word "tonus." Rather we speak of it as adopting a posture suited to the volume of the object it grasps. The case of the viscus is analogous; the bladder assumes postures suited to the volume of its contents. With 50 c.c. of content the bladder assumes what might be termed its 50 c.c. posture; with 200 c.c. contents it adopts a 200 c.c. posture. The muscular wall must in the latter case be on the average twice as long in any direction as it is the former, but such extension of it is, as shown by Mosso and Pellacani, not usually accompanied by anything more than a slight rise in its tension. The *dead* bladder has no property such that when its radius is doubled the wall-tension is hardly increased. The living muscle of the wall could exhibit such a property if, in order to double the radius, it actively relaxed from one contractile form to another, that is, if its muscular fibres actively lengthened from a shorter form which they previously maintained. But here a difficulty arises in view of the prevailing conception of muscular contraction. The notion of tension is attached as a concomitant of shortening and supposes that with greater shortening there must run greater tension. But it has become clear from recent observations, mentioned above, that skeletal muscle in postural reflex contraction may alter the length of its fibres very considerably with little or no change in the tension the muscle exerts. If we could imagine the visceral muscle of the bladder wall replaced by an appropriately shaped arrangement of living skeletal muscle acting posturally under reflex innervation it, as we have seen, could fulfil the requirements which Mosso and Pellacani's observations show that it does fulfil. We may apply the term *posture* to this property of the bladder by virtue of which it solves the problem of acting as a reservoir for quantities of fluid of very varying volume from one occasion to another without allowing the intravesical pressure to attain the reflex stimulus threshold height with one particular fluid-quantity only. But if we do, we must attempt a definition of posture in order that the term may be clear.

Posture may be passive or active. The former—for instance, the postures of a dead body impressed on it by gravity &c.—are of course outside this inquiry. Active posture largely compasses the counteraction of those effects which gravitation, &c., produce in the dead body. Active postures may be described as those *reactions* *in which the configuration of the body and of its parts i of forces tending to disturb them, preserved by the activi tractile tissues, these tissues then functioning statically.* T muscle as an executant of movements is so striking that its of venting movement and displacement is somewhat overlooked. movement, whether active or passive, has brought about a cha configuration of a limb—e.g., by flexing one of its joints—an function of the musculature may be to maintain the new con the posture. In doing this the muscle *prevents* movement, it; it then acts statically and, though in a state of contrac no mechanical work whether the tension it develops in thus m its and the limb's new configuration be great or small. J limb assumes, in result of a movement either passively impos or actively executed by it, various configurations and can mai of these with various degrees of tension—for instance, the arm with a 2 kgm. weight in the hand or a 6 kgm. weight—so muscular viscus after partial evacuation, or after introductio content, assumes a new configuration, conformably with th volume of content; and this new configuration is in both ca tained by the activity of the muscle functioning statically. assumption of a new configuration and the maintenance muscular in the viscus just as in the limb, and the essential the muscular reaction exhibited is in the viscus that which i limb. Such reaction in the musculature of the limb is called it is conducive to clearness if in the viscus also it be termec Both are instances of the postural contraction of muscle the relation of the central nervous system to the postur is very different in the two cases. But the essential ident two cases justifies, and is helpfully kept in view by using, su concerning a muscular reservoir viscus as that it exhibits a qu posture, or a half-pint posture, and so on, according to the its contents, and that both in the one and in the other postu exert one and the same pressure on its contents.

The bladder is no isolated instance of a muscular viscus these volumetric postures.

Stomach.—The musculature of this viscus is now known t very differently in its different regions, not only in rumi birds, but also in the cat, dog, and man. From sphin pylori to pylorus it works as a sort of mill performing fo more or less rhythmic movements, whereas in the fundal pc its contractile wall it forms a reservoir or hopper whence the mill draws its pabulum for milling. A functional requirement of the fundal reservoir is that it should receive easily and accommodate easily food arriving often in large volume quickly, and that it should do so without ensuence of any large rise of pressure. High pressure in the fundus would mean difficulty of entrance of further food from the œsophagus and the premature falling off of one of the factors essential to appetite (Cannon and Washburn [10]), if not the appearance of gastric uneasiness and pain. The mechanical problem before the fundus portion of the stomach is therefore in one way like that before the urinary bladder, it has to alter its capacity considerably without altering greatly its tension; in one respect, however, its problem is the reverse of that of the bladder, it is rapidly filled and relatively slowly emptied.

That the gastric fundus does in fact meet these requirements is shown by actual observations. Thus, in an etherized dog the stomach, exposed and freed from abdominal pressure by suitable incisions of the abdominal wall, showed during twenty minutes an intra-gastric pressure varying between 4 cm. and 5 cm. water, the intra-jejunal pressure at a distance of 40 cm. below pylorus being 2 cm. to 4 cm. water. Both stomach and jejunum at the time contained portions of a previous meal. Then 400 c.c. water were put into the stomach through a cannula. The intra-gastric pressure at once rose, but only by 1 cm. water—i.e., to 6 cm.—and twenty minutes later it had fallen to 4 cm. and the jejunal was then 3 cm. Absorption of water from the stomach is slow, especially under narcosis; nor was there evidence that the fluid had left through the pylorus. The organ had made room for the additional content and exerted on it only the same light pressure as it had done on the smaller content it had held previously. Its muscular coat had exchanged its previous 100 c.c. posture for a 500 c.c. posture practically without change of tension.

Direct observation shows the ability of the fundus to adjust suitably its postural contraction in the converse case of diminishing content. In a man with gastric fistula, by reason of a cicatricial stricture of œsophagus, Kelling obtained the following data. Water to the quantity of 200 c.c. and air to the volume of 300 c.c. were introduced into the empty stomach. The intra-gastric pressure a minute later was 10 cm. of water, but two minutes later had fallen to 8 cm., and it remained at that pressure during the next twenty minutes. It was then found that the water had disappeared from the stomach and also some of the air, 200 c.c. of the latter being all that was withdrawn.

The stomach had got rid of more than 300 c.c. of its contents without any fall in the intra-gastric pressure. The muscular coat had by a "shortening reaction," to use the term adopted above for skeletal muscle, adjusted its capacity, or, expressing the change in a linear term, shortened its contraction length without appreciably lessening or increasing its contraction tension.

The same phenomenon is exhibited by the excised surviving stomach (cat). An expansible bag, whose volume can be increased by injection from a pressure reservoir outside, is placed within the excised stomach. The intra-gastric pressure being 1·5 cm. water, the bag is then rapidly dilated within the stomach so as to raise the intra-gastric pressure to 12 cm. water. The gastric pressure soon falls to the original level again. Similarly, after a rapid injection of water the fundus portion expands (the pyloric does not) and the pressure which rises at the time of injection soon falls to what it was before the injection (Sick and Tedesco [38]). These reactions to distension the dead stomach does not give. Distension of the dead viscus to 200 c.c. causes an intra-gastric pressure higher than that attending distension to 600 c.c. capacity in the excised but still surviving organ. With the living organ *in situ*, and with its circulation intact the difference is greater still. Thus, a small cat took at a meal 150 grm. meat with 150 c.c. milk, the intra-gastric pressure rising to 9 cm. water. The animal was then killed, the stomach taken out and emptied; the introduction of 300 c.c. of fluid into it then caused the gastric pressure to rise to 80 cm. water. Cannon and Lieb [9] have shown that each passing of the cardia by swallowed food is accompanied by a rapid small dilatation of the fundus, and that this dilatation is a reflex operated through the vagus. The gastric pressure at this moment drops a little and then returns to what it had been previously.

As to the relation of the nervous system to the postural contraction which determines the capacity of a hollow muscular viscus, evidence emerges from some of the observations on the stomach. Sick and Tedesco have shown that this postural power is, to an unmistakeable extent, still retained by the excised stomach surviving in a bath of oxygenated Ringer's fluid kept at 38° C. And some observations, as yet unpublished, by Miss S. C. M. Sowton, indicate the same thing. The isolated viscus carefully shielded from changes in temperature was tested for its reactions to increments of content. The increments were introduced into the organ at its own temperature, and under pressures not much above that already obtaining in the organ at the time. After lying locked up in the organ, by closure of the introduction tap, for a brief time, the increment of content was found to be retained in whole or in part by the organ on reopening the tap, and at a pressure often not appreciably different from that obtaining before the increment was added. But the power to adjust the postural contraction of the fundal portion to increased or diminished volumes of content without much change of pressure is, although thus present in the excised organ, less ample and less perfect than under natural conditions of the living normal organ *in situ*, with circulation and nervous system intact.

Among the invertebrata are forms which may be likened to the hollow viscera in so far that they may be regarded from our point of view as muscular bags. Sipunculus, the marine Gephyrean worm, about the size and shape of a cigar, is, so to say, such a muscular bag. Its front end lengthens to a tube, and can be extroverted and then withdrawn again, and this is a normal action when the animal bores the sand. If the hinder part, the muscular sac, be removed, tied to the lower end of a vertical glass tube filled half-way up with water, and if the glass tube and muscular sac be then plunged into a bath of sea-water, the preparation reacts as follows: The meniscus of the fluid in the glass tube settles at some point above the water-level of the bath. There is thus a certain excess of pressure in the bag over and above the fluid pressure of the bath outside it. If then the tube and preparation be plunged deeper in the bath the muscular bag becomes smaller and the height of the water-column in the glass tube still retains practically the same excess above the water-level of the bath. Conversely, if the tube and preparation be raised so that the preparation lies shallower in the bath, the size of the muscular bag increases, but the water-level in the manometric tube soon settles to practically the same height above the water-level of the bath. When the tube and preparation are raised there is, of course, a rise of hydrostatic pressure of the fluid inside the bag in reference to the water-pressure of the bath exerted on the outer face of the bag, and so conversely there is a fall when the tube and preparation are lowered farther into the bath. The muscular bag evidently actively assumes various sizes to suit the different quantities of water thus injected into or withdrawn from its interior, but it exerts practically the same pressure, maintains the same tension, whether it is holding more content or less.

IV.—MUSCLES OF THE CIRCULATORY SYSTEM.

Before leaving the unstriped musculature it may be in place to inquire what other examples of its tonus are interpretable as posture in the same sense as with the bladder and the stomach. Examination of the intestine seems to have been too little prosecuted (from this point of view) to furnish interpretable data. With the circulatory muscular apparatus, however, there are more indications. Unstriped muscle, like skeletal muscle, evidently functions for two main purposes which in some ways it is possible and desirable to consider apart. Of these one is the performance of movements which overcome resistances by the development of tension; the other is the adjustment of contractile length without necessary alteration of mechanical tension. In the muscular mechanism of the circulation these two functions seem to reach high expression in separate portions of the apparatus. In the heart the movement function, the beat contraction, predominates even more than in the bladder and stomach; and as in their case the organ is evacuated by its means. A further resemblance between heart and stomach is that just as the fundal portion of the latter is an expansible reservoir collecting provender for the strong rhythmically contracting pyloric press, so in the heart the auricles are a distensible reservoir which collects the blood, filling the strong rhythmically contracting ventricular-pump. It is of the reservoir portion, the auricle, that postural function, tonus, would be expected rather than of the ventricle, and in some cases, as Fano [13] and Botazzi [6] have shown, tonus phenomena are very obvious. In the tortoise auricle, apart from beat contractions, the capacity of the chamber varies considerably at various times according to its "tonus"; and W. T. Porter [34] has drawn attention to the independence of the tonus, or state of posture as I would prefer to style it, on the one hand and of the beat contraction on the other.

In the blood-vessels as contrasted with the heart the beat contraction is but slightly developed while posturing, or "tonus" is greatly in evidence. When the fulness of an artery alters for reasons outside events in its own wall, the active posture of its circular coat should, on analogy with that of other hollow muscular organs, adjust itself to the diminished volume with little or no change in tension. The mechanical conditions of a conduit tube under a variable head of pressure, and with varying resistance in its exit, practically preclude experimental test of this in the vessel *in situ*. But the observations of McWilliam [19] and of Kesson [17] on fresh excised [illegible] ngly that such a reaction is present. The surv [illegible] under almost the same wall-tension considerably different diameters—i.e., circumferential lengths, capacity postures of its wall. The compensation of arterial pressure when the body is changed in position from the horizontal to the erect is, as is well known, accompanied by a change in the diameter of the arteries. This adjustment of postural capacity to a smaller content, enabling the settling down to that smaller content without appreciable diminution of their grip pressure, is analogous to the adjustment of capacity-posture in the bladder and the stomach. So also the converse reaction, when the erect position of the body is exchanged for the horizontal, finds the arteries able to meet it by postural adjustment to a new and increased postural capacity without necessarily greater tension of the arterial wall. It may be that only when the alterations of filling exceed moderate limits does the stress fall completely upon the pure elastic elements of the wall. When the arteries are examined from a freshly killed animal the arteries are found in a state of contraction. And this persists when the excised portions of them are washed out with oxygenated Locke's fluid. The excised portions are perfectly excitable to break-shock stimuli. This contracted state of the surviving arterial wall suggests that when at death the arterial tree became partially emptied by its contents running over into the venous system the postural action of the arterial wall followed down the diminishing content, just as does the bladder or stomach wall when those viscera are partially evacuated. Meyer [23] found that the best way to restore the arterial wall of the excised piece of artery to its normal calibre was to stretch it under an appropriate load for a quarter of an hour, and that in this way it resumed an approximately normal capacity and retained it after the correcting load had been removed. It will be seen that this is practically the same thing as what has in this article been described both for skeletal muscle and visceral muscle as the "lengthening reaction," although with the difference that in the case of skeletal muscle the reflex arc of the muscle, the proprioceptive arc, is necessary for the reaction, though in the case of the excised artery no such arc is required.

Bayliss [1], however, has demonstrated that the living artery reacts to a sudden rise of internal pressure by contracting, and this observation seems at variance with the above. Bayliss's reaction, I would suggest, is comparable with that contraction of a skeletal muscle which in the spinal preparation can be evoked by suddenly stretching it—e.g., by the fall of a weight attached to its tendon. The extensor of the knee, although it exhibits the lengthening reaction to a gradual though rapid stretch, yet gives the knee-jerk to a sudden tap or quick brief pull. I cannot but think that to a more gradual distension the living artery would give a reaction of relaxation such as the bladder and stomach exhibit under similar distension. The curves given by McWilliam [19] and Kesson [17] showing the reaction of surviving arteries appear to me to support such a view.

Osborne [29] remarks, "It is a mistake to describe the flow of blood in the systemic arteries as a flow of liquid in elastic tubes. Such is certainly the case only when the muscle is fully inhibited or killed. To describe the circulation as occurring through a system of muscular tubes with some elastic tissue aiding the muscles would be more accurate." How true this is is evident from the McWilliam and Kesson experiments. The difference between the effect of adrenal extract on the excised strip of artery according as the vessel has been previously stretched or not illustrates the same point.

The upshot of the foregoing experiments on unstriped muscle is that this type of muscle, besides producing movement contractions or beats, is able to maintain itself at various lengths exerting under all those various lengths approximately one and the same tension. The wall of a hollow viscus is thus able to hold the fluid contents of the viscus at approximately the same pressure whether those contents are copious or not, because of the ability of its fibres to exert the same tension whatever the form, shorter or longer, within a certain wide range, which they have assumed. And their differences in length during this activity can be so great as to allow differences in the capaciousness of the organ suggestive of actual slipping of the muscle-fibres upon each other. Tonus as applied to such a condition seems an equivocal term. If the pressure of the wall on the viscus content be taken as criterion of tonus, then tonus has nothing to say about the state of shortness or of elongation of the muscle, for these are independent of the pressure. If tonus be transferred from its literal meaning to one descriptive of form, and be used to indicate the state of shortness of the muscle—thus, if the bladder when maintaining a small or restricted volume be considered to have more tonus than maintaining a large and "dilated" capacity—then since these states of size have no constant relation to states of tension, tonus appears a misnomer, since it then retains nothing at all of its original and literal meaning. And skeletal muscle exhibits exactly the same properties as these just described in unstriped muscle, and skeletal muscle exhibits them when it performs one of its chief functions, namely, the maintenance of posture. With the unstriped muscles of the viscera and blood-vessels just as with the striped muscles of the skeletal frame, it seems therefore preferable, because more direct, to speak of this form of activity as postural.

The application of the term tonus to sphincter muscles illustrates its employment as meaning posture. A sphincter is described as exhibiting tonus when it maintains a closed posture of the orifice it guards. Tonus here means nothing but postural contraction.

V.—General Remarks.

Finally, we may attempt to examine what are the main characters of the action of muscle functioning posturally.

Reflex postural contraction is characterized by (1) the low degree of tension it usually develops; (2) the long periods for which it is very commonly maintained without obvious fatigue; (3) the difficulty of obtaining by artificial—e.g., electrical stimulation—reflex contraction at all closely simulating the postural contraction produced by the natural stimuli whatever these may be; (4) by the relative ease with which reflex inhibition interrupts the postural contraction, and by the (5) "lengthening" and (6) "shortening" reactions obtainable from muscles exhibiting postural contraction. In regarding these features as distinctive various reservations have to be made. As to the first feature the tension strength, though commonly quite mild, is sometimes quite powerful. For instance, in the decerebrate cat the postural tension of the anti-gravity muscles, though usually just sufficing to support the superincumbent weight of the animal in its erect posture, may sometimes be far greater than that, both when the preparation is on its side or supine—i.e., inverted. Magnus and de Kleijn [20] have pointed out that the reinforcement of the postural extension of the limbs by influences from the labyrinths is greatest in the inverted position of the preparation. This might be taken as an indication that the reflex posture of the preparation is not really that of standing. But, as is well known, many animals when placed supine throw up the limbs into an attitude of full extension. The accentuation of the postural extension when this is done in the decerebrate preparation is, therefore, merely the reflex assumption of a defensive posture of common occurrence, and does not argue that the postural action of the extensors in the right-side-up attitude is not just as truly reflex standing.

Of more interest is the fact that when the reflex postural contraction is very powerful it seems to pass over into the full sthenic reflex action which can be produced by ordinary artificial stimulation of an afferent nerve appropriate for reflexly exciting the muscle. The lengthening reaction cannot be obtained from a muscle in ordinary sthenic reflex contraction as excited by the usual faradic stimulation of an afferent nerve; nor can this reaction be obtained, in my experience, from a posturally contracting muscle if its postural contraction is very strong. In so far then the reflex postural contraction seems when very intense to pass over into and become indistinguishable in this respect from ordinary reflex contraction such as occurs in "movement" reflexes, &c. Again, though reflex postural contraction is in many cases inhibited by stimulation of any appropriate afferent nerve with an ease and delicacy of grading truly remarkable there are instances where even powerful inhibitory stimuli fail to inhibit it. Such instances occur in certain phases of strychnine poison which augments postural contraction; also in the contraction of the fore-limb muscles in the embrace-posture of the male frog and toad at the breeding season.

It would seem, with regard to the skeletal muscles of vertebrates, that the maximal tension which the muscle can maintain posturally is distinctly less than the maximal tension which it can exert when employed for executing movements, and that when tension above that required for ordinary postures is demanded it is obtained by the same kind of contraction as that employed for movements; and that this latter kind of contraction cannot be long maintained owing to fatigue, whereas the kind of contraction maintaining ordinary postures which require less tension can be long maintained without fatigue.

The third feature characteristic of postural contraction is its relative unfatiguability. The unfatiguability is often extraordinarily great. The postural contraction of the extensors of the limbs in the decerebrate cat may persist not only for hours, but for days. I have seen it continued as far as my opportunities for observing it went, apparently without complete intermission for six days. The extremely powerful embrace-posture of the male toad continues without remission for many hours. The postural contraction of the muscle which keeps the shell of pecten shut was observed by Parnas [30] to withstand a load of 3 kilos, in addition to the pull of the elastic ligament which opens the shell, for three hours. An oyster removed from the water will keep its shell valves closed against the spring of the elastic ligament for thirty days on end. The resistance to fatigue exhibited by the muscles in cataleptic postures is well known.

It may be argued that the mild intensity of the contraction maintaining posture is the explanation of this relative immunity from fatigue exhibited by postural as compared with ordinary contraction. But, as some of the above instances show, the intensity of the postural contraction if gauged by the load which the contracted muscle can counteract is sometimes not slight.

There is another possible, and probable, reason for the unfatiguability of postural contraction. A skeletal muscle when at rest, that is, when it is not being stimulated, exhibits a certain length. It is extensile and elastic; when loaded with different weights it has under each weight, over a certain range, one definite length; as the weights are changed its length changes from that proper to one weight to that proper to another. The tensile strain of the muscle varies proportionately with the weight, so that we may say that each particular grade of tensile strain gives the muscle a particular length. When the muscle is stimulated it assumes a different and shorter form. But its tensile strain and its length are still related; over a considerable range of weights and strains the length of the contracting muscle is longer under a heavy load than under a lighter one. Further, for the muscle to assume by contraction any particular degree of shortness the excitation has to be stronger when the load is greater than when the load is less. So that tension here is the measure of the intensity of action of the muscle. Other measures of the intensity of activity of the muscle are (1) the amounts of mechanical work and heat produced by the muscle, and (2) the amount of chemical change undergone by it.

A single stimulus, that is to say, a stimulus such as a single induction shock applied to a motor nerve, which evokes a single nervous impulse as it is known to do, causes a muscle to contract with that brief contraction called technically a twitch. The usual view of the nature of any contraction lasting longer than that is that it is due to further ensuing stimuli which cause further similar twitches, and that these latter fuse with the precurrent one. The twitch is a process considerably expensive of energy; the energy appearing in the form of mechanical work and heat, and coming from chemical compounds degraded in the process of contraction. With a fused series of twitches, a tetanic contraction, the muscle usually shortens against some load till it reaches a certain grade of shortening, and this grade of shortness is then, within limits, maintained so long as the stimuli continue to be applied. Then, on withdrawal of the stimuli, the muscle lengthens under the pull of the load, returning to its original resting length. In such a case it is, of course, only in the early part of the tetanic action—that is, when the load is actually being moved—that work in the mechanical sense is performed. When the actual moving of the load ceases, and the load is merely being retained in the new position into which it has been lifted, the muscle, although counteracting the load, is doing no work in the mechanical sense. In the former period of the tetanic contraction the muscle does external work; in the latter it does no external work. But this latter "static" period of the contraction is nevertheless expensive of muscular energy. The muscle continues to exhibit a considerable turnover of energy. It is then, it is true, playing a rôle which is merely that of an elastic band or ligament, not inexpensively, however, as to energy-cost and metabolism, as a ligament would, but at the cost of considerable energy transformations. The muscle, however, offers the advantage of supplying an elastic structure which can modify its length and elasticity according as this or that purpose is required of it. The determination of the amount of the energy-turnover in tetanised muscle thus acting statically can be estimated by measurement of the heat production or of the oxidation products. Such observations (Bernstein and Pober, Johannson) show that the energy-cost increases with the load which the contraction counteracts, and its increments increase more quickly than the increments of load. Also that the energy-cost per unit of time per kilo of load increases as the period of the contraction is prolonged. Further, in the Stockholm laboratory, it was found that the energy-cost increases rapidly with increase of the degree of shortening which the contracting muscle maintains.

As to the actual figure to which the cost in a typical example works out, Parnas, on the basis of a table of results by Johannson and Koraen, arrives at the following for the flexors of the elbow in man; 2·5 mg. CO_2 per second for a 49 kilo load, and 8·3 mg. CO_2 per second for a 160 kilo load. Such a cost accords with the view that the muscle, in thus supporting a load which it has lifted, is continuing a process essentially the same as that underlying the lifting period when external work is done; in short, that it is continuing in an additive way essentially the same process as that underlying the twitch. And it is not surprising hat such a process should entail, when prolonged, relatively rapid signs of exhaustion or fatigue.

But when we turn to inquire the energy-cost of some postures as measured by the chemical amount of oxidation products which they entail and by the less measurable but perhaps equally significant speed of onset of signs of ensuent fatigue we find a discrepancy between the cost of these postures and that of prolonged ordinary tetanic contraction. Parnas, as the result of experiments on the postural action of the adductor of the shell of molluscs above described, shows that a weight can be supported by the muscle acting posturally with only a trifling increase of the oxygen intake or carbon dioxide output. He calculates the amount of CO_2 which would be given off by a frog's muscle holding up by tetanic contraction the same weight for the same time. He concludes that 10,000 times more energy will be expended by the frog's tetanically acting muscle than by the molluscan muscle acting posturally. According to him the experiment of Johannson and Koraen reckoned for a load of 3 kilo gives a cost of 403 mg. O_2 per hour; whereas with pecten he found where the adductor supported 3 kilo the consumption of O_2 per hour was 0·008 mg. It has to be remembered that the mass of muscle concerned in the human case was very much greater than in the case of pecten, although that in some ways only makes the contrast the more remarkable. The factors involved in the comparison are, it must be admitted, extremely difficult to assess.

H. Meyer and Alfred Fröhlich [22] noted that the enduring contraction produced by tetanus bacillus involves an extremely low metabolism, the glycogen content even increasing in the rigid muscles.

Roaf made observations on the CO_2 output of the decerebrate cat during prolonged postural rigidity of its "standing" musculature. He found the CO_2 output in this condition not detectably greater than during complete muscular paralysis by curare. Bayliss also has found the chemical turnover accompanying tonic contraction of skeletal muscles extremely low, although distinctly above that of the muscles when at rest.

The facts tend to show that in many cases postural contraction is astonishingly economically maintained; that the turnover of chemical energy involved by it is extremely low. In such cases its relative unfatiguability may well be related to its economy of maintenance. So strikingly does this aspect of it contrast with the expense of maintenance and the relatively rapid fatiguability of ordinary tetanic contraction as to suggest that the chemico-physical process underlying postural contraction is in part at least essentially other than that underlying twitch contraction, and the fusion of twitch contractions termed tetanic contraction.

The supposition has been put forward that in maintaining this economical postural contraction the muscle-fibre, or some part of it, clots, changes from *sol* to *gel.* For instance, that with pecten, the glassy adductor, the postural adductor, when, under the approximation of its attachment points, as the valves near each other by the action of the movement muscle, it has become shorter and thicker, "clots," changes to a gel, becomes semisolid, retaining that configuration which it had at the moment when it entered into the solid phase. The posture of closure of the shell valves is then maintained, without further expenditure of energy, by the mere elasticity of this new-formed solid connexion between them. The clot or gel it must be supposed can be dissolved by nervous influence reaching the muscle. When this happens the contents of the muscle-fibres melt again and the muscle relaxes, the shell valves opening apart under the pull of their elastic ligament. On this view evidently one of the nerves supplied to the postural adductor can cause the contents of its muscle-fibres to gel, to solidify; another of its nerves can cause it to unclot.

And the postural adductor can also contract in the sense that it can shorten itself; but this shortening proceeds more sluggishly than does that of the movement adduction, whose fibres are striated.

Such a view is easily applicable to visceral muscle such as vesical and gastric, and to that of the blood-vessel wall. In extending it to vertebrate skeletal muscle points of modification would be the following:—

In them postural contraction, just like other contraction, is under natural circumstances only present in response to nervous impulses reaching the muscle from the motor neuron. A number of observations show that in postural contraction the muscle exhibits action-currents succeeding each other at rates varying with different muscles and under different circumstances from 40 per second to 80 or 90 per second. The maintenance of a gel state would, on the assumption that it underlies postural contraction, depend, therefore, on the receipt of nervous impulses, without which the particular part of the muscle-fibre which has solidified reverts to the fluid phase practically at once. And to these muscles no peripheral nerve-fibre is distributed stimulation of which unclots the muscle; the postural contraction in them can be readily suppressed by nervous action; in terms of the hypothesis they can be readily and at once unclotted by nervous action, but that is always brought about by suppressing the motor neuron discharge that is necessary for the maintenance of the gel. The resolution of the gel is therefore effected indirectly by afferent nerve-fibres, or connector nerve-fibres from other nerve-centres, which inhibit the motor neurons which promote and maintain the gel. Also, since vertebrate skeletal muscles are obviously capable both of executing movements and maintaining postures, the question arises, in following the hypothesis, are some of the fibres of the muscle specialized for postural contraction and others for non-postural contraction? Do some solidify under nervous influence and some contract? Or do some or all of the individual fibres possess in themselves both mechanisms, a mechanism for ordinary contraction and a mechanism for resisting tensile strain by becoming semisolid? There is the theory developed by Botazzi that in the muscle-fibre the sarcostyles are the contractile mechanism, and the sarcoplasm the tonic mechanism, or, as I would prefer to say, the postural mechanism. On the view now discussed the sarcoplasm therefore would be the coagulable or solidifiable element. The two diseased conditions, congenital myotonia and myasthenia gravis, suggest forcibly that the functional mechanical activity of muscle is twofold in nature, tonic and non-tonic, postural and non-postural. Farquhar Buzzard [8] has treated of the applications of the "functional duality" theories of Grützner and Botazzi to clinical phenomena and problems, and has shown how full of suggestion such a theme can be made. And it may be recalled that Perroncito [33] (1902) asserts the presence of two kinds of motor-endings in the striped muscle, one the well-known motor-plates of medullate nerve-fibres, the other an ending belonging to non-medullate nerve-fibres reaching the muscle; and Boeke [4] confirms such a view. Mosso [25] (1905) put forward the suggestion that the latter endings are for tonic contraction and from the sympathic. De Boer has recently ascribed the Brondgeest phenomenon of the tonus of the flexors of the frog as due to severance of sympathetic fibres passing through the sciatic nerve-roots, and suggests that the cerebrospinal motor nerves control the carbohydrate metabolism, the sympathetic the protein (tonus) metabolism of the muscle. Pekelharing [31, 32] has indeed shown that prolonged tonic action of the skeletal muscles, as in the reflex "standing" of the decerebrate cat, is accompanied by an increase of creatin in muscle and creatinin output.

Even where, as in Pecten and Echinus, the muscle-cells for movement contraction and postural action are so specifically differentiated, the view does not necessarily imply that the process of contraction in them is wholly different. Though the twitch contraction of a frog's muscle isotonically recorded retains its new and shortened form hardly at all, the twitch of a tortoise muscle retains it for a fifth of a second.

In the twitch of the bladder muscle it is retained for much
Such authorities as Fick and Hermann have suggested analogy b
the contracted state of rigor mortis, known to be due to clotting
muscle-fibres, and the contracted state in the twitch and in t
contraction. Such considerations indicate that what in postural
is a long enduring clot phase may also be present in movement
though in these as a very brief phase.

Biedermann [3] (1904) wrote: "Tonus of muscle is a s
enduring shortness which only in part depends on enduring exc
by nervous centres, and for its other part is produced by those
processes in contracted muscle which otherwise cause relaxati
lengthening, being either not at all or only incompletely present t
the alterations brought about in the muscle by the precurrent excit
Parnas [30] (1910), after criticizing the want of clearness often
ing to the conception of tonus—he excepts, however, Biede
above quoted sentence—offers the following: Tonus is every sta
state of a normal muscle in which for each given length—less th
longest occurring in the unstretched muscle—the turnover of
is independent of the load. Tonus muscles are those muscles
throughout the whole range of lengths, which extends betwe
longest and the shortest lengths occurring physiologically, can
at one unaltered length both when under tension (loaded) or not
tension, and maintain that length without increasing their energ
over. He proposes length as a measure of tonus, thus, if L is the
of the muscle in its shortest tonic length, and l its tonic len
observed on a particular occasion, $\frac{L}{l}$ is the measure of the tonus
that time. Thus if gastrocnemius be 30 mm. long at shortest
tonic length at a given time is 35, $\frac{30}{35} = \frac{6}{7}$ T.

These definitions are of interest here because they atten
describe a kind of activity of muscle unlike that usually ac
as the universally typical one, namely, the tetanic contractio
duced by faradizing the muscle or its motor nerve. The naïv
of postural action would regard it as a mild degree of or
tetanic contraction. It has been the object of the present art
put forward and examine the suggestion that the kind of acti
muscle which the above definitions attempt to describe is that of
when functioning in posture, and that it extends not only
posturing of the skeletal musculature but to that of the viscer
vascular musculature as well. A prime feature of this activity
in it the length and mechanical tension of the muscle are va

largely disconnected one from the other, and that any required shortened form of the muscle may be maintained with much greater economy of metabolism than in ordinary tetanic contraction, and with great relative freedom from fatigue.

In a previous contribution to this Journal (1906) [37] an outline was attempted of what was there termed the proprioceptive division of the nervous system. That division, it was shown, has distinctive features anatomical and functional, rendering advisable its consideration as a mechanism with peculiarities sufficiently its own to warrant its being dealt with broadly as an entity by itself. The postural action of muscles and nerves, the subject of the present paper, is a main outcome of the functioning of the proprioceptive part of the nervous system, at least it is so as regards the skeletal musculature, perhaps as regards the visceral and vascular musculature also. Reflex maintenance and adjustment of posture is a chief portion of the reflex work of the proprioceptive system, just as sensation of and perception of posture is a chief portion of the psychical output of that system.

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ON MUSCLE TONUS.

BY J. W. LANGELAAN

Amsterdam.

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CHAPTER I.—THE ATONIC MUSCLE.

(*A*) *The Load Variable.*

These researches are based upon the conception of a simple proprioceptive reflex arc upon which the tonus depends. As soon as this arc is broken the muscle becomes atonic. My experiments on the atonic muscle have only been made with the M. gastrocnemius of the frog. In the beginning I cut through the motor nerve, but because it was not excluded that motor fibres might also enter the muscle with the blood-vessels, I took the muscle entirely out of the body and suspended it in a moist chamber. When the muscle is not compelled to do active work and the experiment does not last too long, the suppression of the blood circulation seems to have no sensible influence upon the elastic properties of the frog's muscle.

The units adopted in this part of the research are: 1 second as the unit of time [T]; $\frac{1}{10}$ mm. as the unit of length [L, l]. The enlargement by the lever of the length-recorder amounted to 5·65, so that the real unit of length is $\frac{1}{56\cdot5}$ mm.; 1 gramme as the unit of weight. The derived quantities are: The increment in length of the muscle per gramme increase of the load $\left[\frac{\Delta L}{\Delta P}\right]$. I only use this quantity under the condition that the elastic after-phenomenon is eliminated, in which case $\left[\frac{\Delta L}{\Delta P}\right]$ represents the *elasticity* of the muscle; the increment in length of the muscle per second $\left[\frac{\Delta L}{\Delta T}\right]$, when the load is constant, or the influence of the variation of the load upon the value of this quantity, is eliminated. Under these conditions the quantity $\left[\frac{\Delta L}{\Delta \tau}\right]$ represents the after-lengthening or the after-shortening of the muscle. Because only plastic bodies show these after-phenomena, I propose to consider the quantity $\left[\frac{\Delta L}{\Delta \tau}\right]$ as a measure of the *plasticity* of the muscle. There are, of course, other criteria of plasticity, but the after-phenomenon seems to me the most simple and most valuable.[1]

Elasticity and plasticity are allied phenomena, but they differ in many fundamental respects. Elasticity may be defined as momentary yielding to a sudden stress, plasticity as slow yielding to a continuous stress. Elasticity is a reversible phenomenon, because the strain disappears entirely with the removal of the stress; plasticity is an irreversible one. After a prolonged stress the body does not return to its original state, but tends to a state different from the first. The variation induced in the body is therefore a permanent deformation. The property of showing definite permanent deformations under the

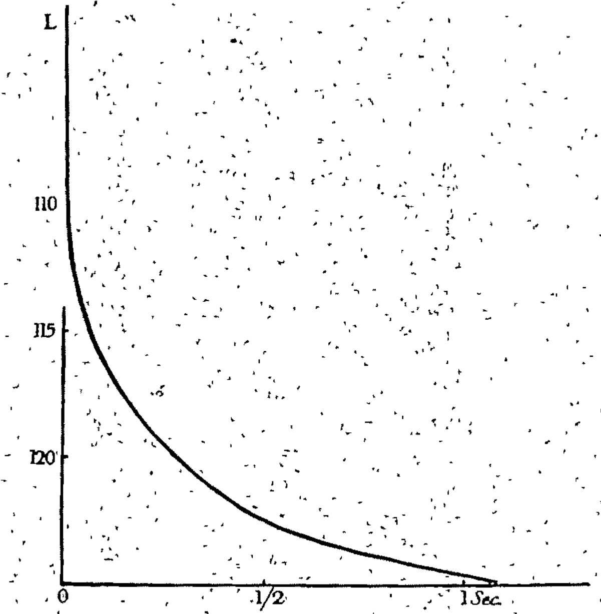

FIG. 1.—Reproduction of the beginning of the extension curve of Experiment 1.

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[1] Cf. Noyons, A. K. M., in his dissertation "Over den Autotenus der Spieren," 1908.

influence of external forces of a certain finite duration is the chief criterion of plastic bodies [38].

Experiment 1, *November* 9, 1904.—In this experiment the M. gastrocnemius was cut out of the body at 12.10 p.m., suspended in the moist chamber and charged with 2·5 grm. The length of the muscle under this load was 3·2 cm. At 12.15 p.m. this load was abruptly increased by 22 grm. In consequence of this increase of the load the muscle lengthened, but the surcharge was immediately taken away. At this moment the muscle shortened and returned to its original length. The return to the original length under these conditions was also observed by Blix [1]. The elasticity of the muscle amounted in this

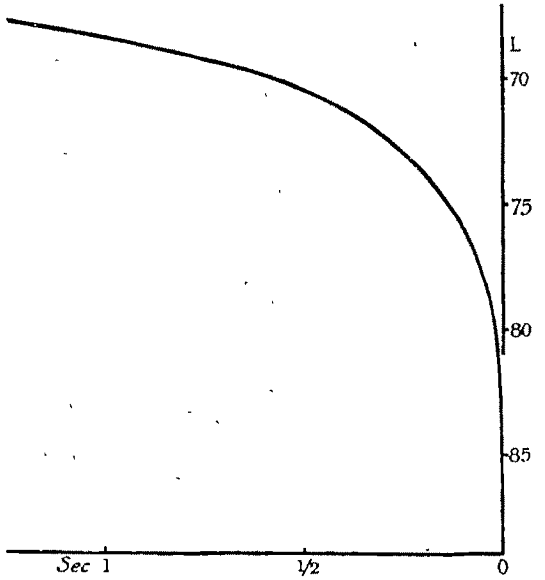

FIG. 2—Reproduction of the beginning of the unloading curve of Experiment 1

case to 4·55. Again the charge of the muscle was increased by 22 grm., but now this surcharge was left on the muscle for 51¾ minutes. Then the surcharge was taken away. Table I gives the result of this experiment [illegible] After a sudden elongation of about 109 units, the muscle cont[illegible] to lengthen, and after 51¾ minutes this after-lengthening [illegible] finished. Figure 1 is the reproduction of the beginning of the [illegible] and we see that the initial elongation passes continuously into t[illegible] lengthening. For this reason it is not possible to [illegible] accuracy the elastic elongation, and hence I choose the [illegible] 0 + ϵ, ϵ signifying a short, not quite determined interval of ti[illegible] the same reason a mark of interrogation is placed behind the first number (109) in the second row. This notation indicates that in a short interval of time (ϵ) an elongation takes place of 109 units, and that this elongation is approximately the elastic lengthening of the muscle under the influence of the surcharge of 22 grm. Fig. 2 reproduces the unloading of the muscle from the surcharge, and we see that here also the sudden shortening continuously passes into the after-shortening.

In the third row of Table I, I give under $\left[\frac{\Delta L}{\Delta \tau}\right]$ the plasticity of the muscle. We notice that under the influence of the surcharge the plasticity diminishes, but even after 51 minutes the after-lengthening is still going on. The same thing happens after the unloading of the muscle. In Tables II and III, two other experiments of the same kind are reproduced. We conclude from these experiments *that an atonic muscle, loaded with a moderate weight* (20 *to* 30 *grm.*), *does not reach a definite length even in about one hour, but continues lengthening and tends finally to a state of rest.* This state of rest, as I will show later, is in general only a state of apparent equilibrium. It ensues, therefore, from this simple experiment that the length of the atonic muscle is not entirely determined by the load. The experiment proves further that after the surcharge is taken away the muscle returns to a length greater than before.

Table II contains for this purpose an extrapolation, which shows that even five and a half hours after the unloading the muscle would not have recovered its original length. We conclude from this observation *that a continued pull exercised by a moderate weight suspended on the tendon of a muscle produces in that muscle a permanent deformation.*[1]

In the fourth row I give the quantity $\left[\frac{\Delta L}{\Delta \tau} \times \tau\right]$, i.e., the plasticity multiplied into the time elapsed since the moment of loading or unloading. Within the limits of experimental error this quantity is constant. We conclude from this fact *that the after-lengthening and after-shortening are directly proportionate to the logarithm of the* [illegible] *f we take as zero of time the moment immediately preceding* [illegible] *ing or unloading of the atonic muscle.* I found this relation [illegible] ently of the work of Percy Phillips [37], who some years [illegible] d the same rule to hold good for the after-phenomenon [illegible] s.

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[1] [illegible] rrive [illegible] similar result: "Les muscles sont comme le plomb et le beurre qui, [illegible] ont été [illegible] s, conservent indéfiniment l'empreinte qu'ils ont reçu" [33].

Experiment 4, *November* 15, 1904.—In this experiment the M. gastrocnemius was taken out of the body at 10.10 a.m., and suspended in the moist chamber at 10.15 a.m., loaded with 13 grm. Under these conditions the length of the muscle was 3·2 cm.; the experiment began at 10.32 a.m. The régime of this experiment is the following: A light weight-pan of about 13 grm. was fastened to the muscle; in this pan mercury may flow in and out at a constant rate, and in this way a regular rising and falling of the load was attained.

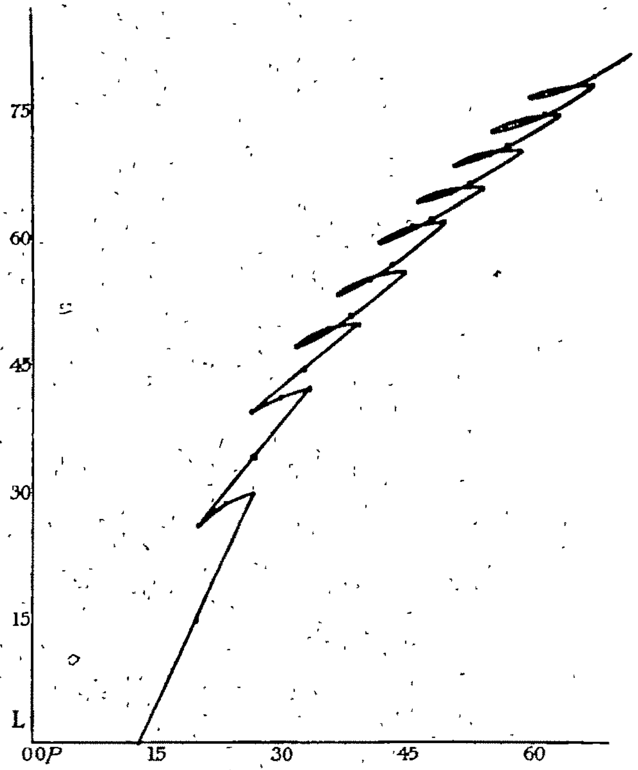

FIG. 3.—Length-load diagram of Experiment 4.

In the third row of Table IV the rate of increase and decrease of the weight is given in grammes per minute. During the course of the experiment the rate of increase, as well as the rate of decrease, varied a little, but these deviations from constancy do not visibly influence the form and the direction of the curves. The duration of the inflow is six minutes, and that of the outflow is three minutes. The total period of nine minutes is usually called a cycle, and the variation of the load during that interval of time a cyclical variation. Because the outflow does not exactly compensate the inflow, there is at the end of each cycle a surplus. In that case we speak of non-closed cycles. The régime is therefore one of non-closed cyclical variations of the load.

Fig. 3 is the graphic representation of this experiment. Each cycle is composed of a rising and a falling load curve, agreeing respectively with a phase of increasing and decreasing load. As a general result we find that the falling curve lies above the rising curve of the same cycle. The meaning of this result is that the increasing load has not only provoked an elastic elongation, but also a plastic deformation of the muscle. The same fact we notice if we compare a falling curve with a rising one of the next cycle. These two curves have only one common point, viz., the first two cycles or the curves form a loop. If the phenomenon was only an elastic one, it would be reversible, and the two curves should agree. Hence the formation of a loop proves that there is also an irreversible part in the phenomenon, and this irreversible part is the plastic deformation of the muscle, caused by the cyclical variation of the load.

Experiment 5, *November* 16, 1904, is of the same kind (Table V, fig. 4). It shows that even after twenty cyclical variations of the load the plastic deformation is still going on. We conclude, therefore, *that a cyclical variation of the load produces in the atonic muscle a deformation, and that this deformation, according to its plastic nature, is at least partly a permanent one.*

We may arrive at the same conclusion in a somewhat different way. If we draw a vertical line in figs. 3 and 4, this line, if it cuts the tracing at all, cuts the curve generally in more than one point; the same happens if we draw a horizontal line. Because along a vertical line the load is constant, and along a horizontal line the length is invariable, we conclude *that the length of the atonic muscle is not entirely determined by the load, and inversely that a certain load does not necessarily agree with a fixed length.* In other words, the length of the muscle is partly determined by the plastic deformation produced by the load, and the amount of this deformation depends upon the way the load reached its actual value. Systems of this kind are usually called systems endowed with hysteresis, and the phenomenon, that the state of these systems is not entirely limited by external forces, is called hysteresis.

Fig. 4 presents another feature of some interest. It shows that the loops, from nearly vertical in the beginning, take on a more and lifting of the load. It follows from this table that the load lifted influences the value of this constant. In order to demonstrate this influence, I divided the mean value of the constant of plasticity in each interval succeeding the lifting of a weight by the value of the weight taken away. The fourth row of Table VII $\left[\frac{\frac{\Delta L}{\Delta \tau} \times \tau}{\Delta P}\right]$

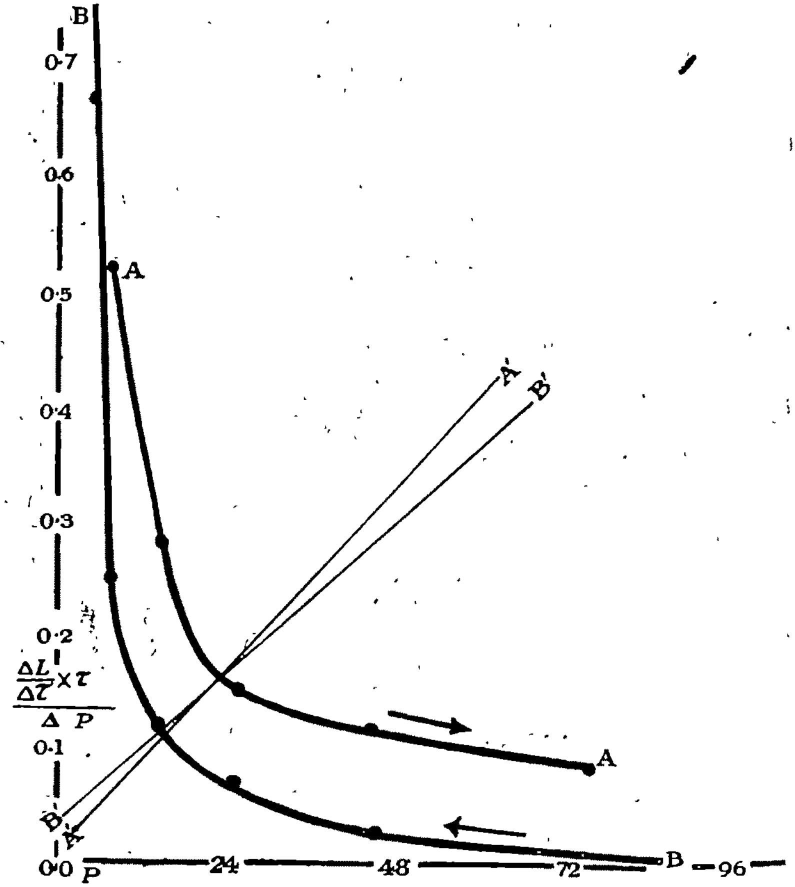

FIG. 6.—Graphic representation of the results of Experiment 7. The reduced constant of plasticity as a function of the charge of the muscle during each period of after-lengthening (AA curve), and during each period of after-shortening (BB curve).

gives these values. In fig. 5 I plotted the value of this constant against the charge of the muscle during each period of after-shortening. The curve which ensues is symmetrical in respect to the axis AA and resembles a hyperbola.

Experiment 7 (Tables VIII, IX) is of the same kind, but in that case I observed the after-lengthening of the muscle after each increment of the load, as well as the after-shortening after each successive lift. Fig. 6 is the graphic representation of the results of the experiments. The arrows along the curves show the direction in which the load was varied. Each of the two curves is symmetrical, but the axis of symmetry of the two tracings do not coincide. Both curves resemble a hyperbola. The curve AA, corresponding to the successive loading of the muscle, lies above the curve of the discharging (BB), which proves that the charging and discharging per saltim of the muscle also produces in the muscle a plastic deformation. The more special conclusions which might be drawn from these experiments are of physical rather than of physiological interest. I conclude, therefore, only *that the value of the constant of plasticity depends as much upon the charge of the muscle as a whole as upon the load which is added or lifted.*

The régime of Experiments 8 and 9 consists in successive series of cyclical variations of the load.

Experiment 8, *November* 30, 1904 (Table X).—The muscle was taken out of the body at 12.40 p.m., suspended in the moist chamber, and charged with 3·65 grm. The length of the muscle under this initial charge was 3 cm. At 1.11 p.m. the load was augmented with 10 grm. The lengthening of the muscle was observed for about twenty minutes. The mean value of the constant of plasticity during that interval amounted to 4·12. Now the weight of 10 grm. was carefully, and without shocks, removed from the weight-pan and immediately replaced in the same way. This procedure means a quick variation of the load from (3·65 + 10) → 3·65, and again from 3·65 → (3·65 + 10). This cyclical variation of the weight was repeated five times in succession; the time necessary for the performance of these five cyclical variations of the load was about one minute. By these five cyclical variations the after-lengthening is set up, but this effect quickly dies away. If we take as the zero of time the moment immediately before the last cyclical variation was terminated, we find for the constant of plasticity the mean value 1·19. A new series of five cyclical variations again activated the after-phenomenon, but the constant of plasticity is only 0·75. The general result is now that each series of cyclical variations of the load exalts the after-lengthening for a short while, but that the constant of plasticity decreases regularly after each series.

Fig. 7 is the graphic representation of Experiment 8. Along the abscissa I placed the ordinal number of the series of cyclical variations; 1 means, therefore, the first series; 2 the second series of five cyclical variations of the load, and so on. Because the cyclical variations were

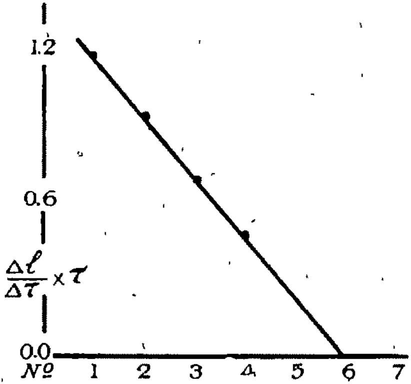

FIG. 7.—Graphic representation of the result of Ex
The constant of plasticity as a function of the ordin
the series of cyclical variations of the load.

that the effect of a series of cyclical variations of the load quickly diminishes, and that probably the effect of the sixth or seventh series would be *nil.* Successive series of cyclical variations, therefore, lead to a state of rest. The next experiment will prove that this state of rest is not a state of true equilibrium.

Experiment 9 (Table XI) is of the same kind. The surcharge amounted in this experiment to 30 grm. The régime was not uniform throughout, but after three series each of five cyclical variations followed two other series of ten cyclical variations. Fig. 8 reproduces the result of the experiment in the same manner as fig. 7. The five points representing the five series of cyclical variations do not lie upon the same straight line, but belong to two different lines. The effect
series of cyclical variations, therefore, depends not only upon the
hich is varied, but also upon the number of cyclical variations
The lines converge, but do not cut the abscissa in the
e final state reached through successive series of
f the same load, therefore, are not states of true
were the case, the lines ought to converge to a
bscissa. We conclude, therefore, *that the final*
muscle under the influence of a constant load
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f that load the muscle approaches slowly to
The length of the muscle in a state of
s the

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increased. This increment, however, was very considerable for the last period. The load was constant during the experiment, and not lifted; hence it is possible to determine the after-elongation during the period of vibration. The broken line in fig. 9 represents the effect of the vibrations upon the value of the plasticity. In reality the line will be fluent, and not broken, but my determinations of the value of $\left[\frac{\Delta L}{\Delta \tau}\right]$ are too scanty to fix more exactly the character of the line. Zigzag lines indicate in fig. 9 the moment the muscle was put into vibration; fig. 10 is the reproduction of the graphic records of the second and of the fifth groups of vibration.

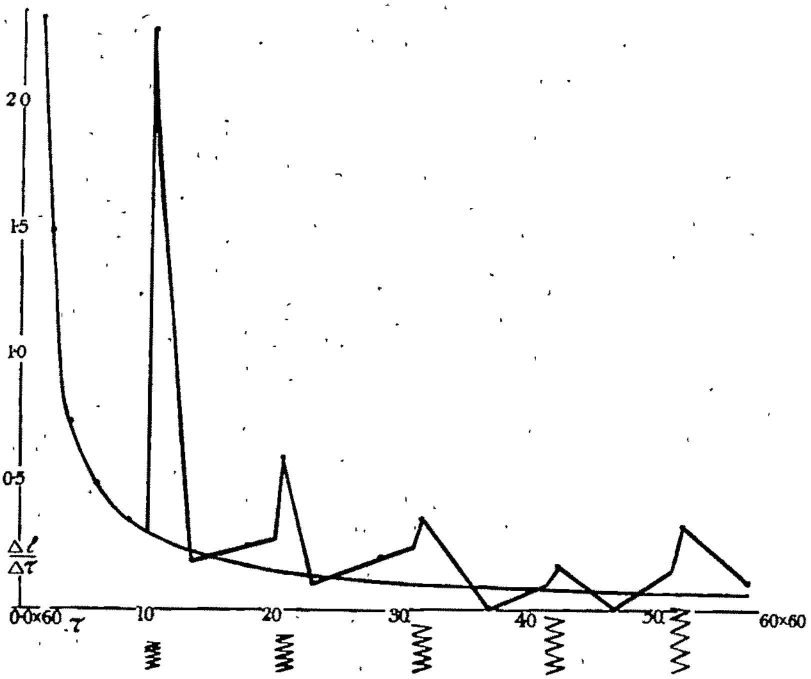

Fig. 9.—Graphic representation of the effect of vibrations upon the value of the plasticity.

The experiment proves that the muscle is extremely susceptible to vibrations, especially at the beginning of the after-lengthening, and that the effect of each period of vibration quickly diminishes. Only the last set of vibrations is followed by a somewhat greater effect. This increment of the effect, however, is caused by a strong increase of the amplitude and of the duration of the vibrations. This experiment is of interest, because it elucidates the cases in which I found at some given moment a constant of plasticity larger than would agree with the relation $\left[\frac{\Delta L}{\Delta \tau} \times \tau = \text{const.}\right]$. A close examination of the tracings in these cases always showed small vibrations. In the tables these vibrations are indicated by a zigzag line. The deviations here and there from the simple logarithmic relation are therefore caused by vibrations. We conclude from Experiment 10, and from the deviations in the experiments already mentioned, *that the most efficacious expedient, to set up the after-lengthening and to reduce the atonic muscle to its ultimate equilibrium-length, is to put the muscle into longitudinal vibration.* The weight with which the muscle is loaded probably influences the course of this process, in so far that the final length is more quickly reached with a moderate load than with a light load.

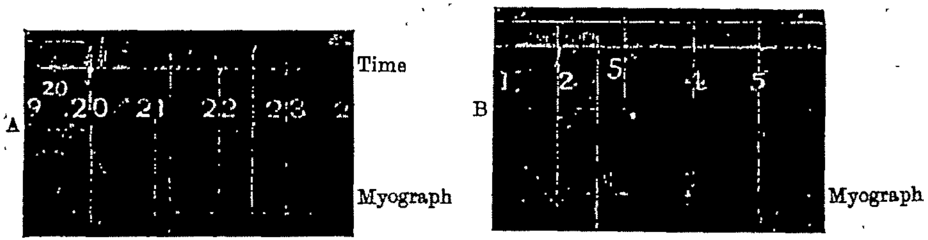

FIG. 10, A and B.—The effect of longitudinal vibrations upon the length of the atonic muscle. Fig. A is the reproduction of the graphic record of the second group of vibrations, and fig. B is the reproduction of the fifth group of vibrations. The enlargement is about ten times.

The phenomena so far described only hold good for moderate charges. The upper limit for the frog's gastrocnemius lies in the neighbourhood of about 70 grm. To prove this I reproduce the beginning of Experiment 11, December 29, 1904 (Table XIII). The muscle was charged per saltim with 74·8 grm. In this case the quantity $\left[\frac{\Delta L}{\Delta \tau} \times \tau\right]$ was not constant but increased with the time. This means that the after-lengthening did not proceed according to the logarithmic rule, but more rapidly. The same phenomenon is shown by soft metallic wires, and in these cases the constant weight ends by rupturing the wire.[1]

In the next group of experiments I have tried to eliminate the after-phenomena. The methods adopted for this purpose are increasing and decreasing the load as slowly as possible, or varying the load very rapidly and repeating this procedure several times. In both cases the curves obtained are extension curves of the atonic muscle, and the difference lies only in the rate of increase or decrease of the weight.

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[1] For the analogy between the atonic muscle and soft metal wires see A. Morley and G. A. Tomlinson, *Philos. Mag.*, 1906, sixth series, vol. xi, p. 380; and E. Lenoble, "Thèses de Bordeaux," 1900, No. 12.

The régime of *Experiment* 12, *October* 31, 1904 (Table XIV) consisted in increasing the weight as slowly as possible. In the third row of Table XIV under $\left[\frac{\Delta P}{\Delta \tau}\right]$ the increase of the load per second is given. I tried to establish a uniform rate, but, as will be seen from the table, this aim was not reached. The rate of increase of the load fell from 14 mg. to 4 mg. per second and then rose a little to 7 mg. The influence of this deviation from the uniform rate upon the form of the tracing is probably not very considerable, because it took place smoothly and without shocks. After two hours the maximum charge of 72 grm. was attained, and from that moment the load was decreased. Very soon, however, the experiment was stopped, in consequence of a defect in the apparatus.

The chief result of this experiment is, that the extension curve of the atonic muscle is nearly rectilineal, i.e., that the increase in length of the muscle is approximately proportionate to the increment of the charge. The deviation from the simple law of proportionality of strain to stress resides in the beginning of the curve and disappears towards the end. Other experiments of the same group prove that the deviation of the extension curve from the straight line becomes more and more considerable by augmenting the rate of increase of the load. We conclude, therefore, *that the extension curve of the atonic muscle is probably rectilineal when the rate of increase of the load is very small, or more exactly, that the extension curve approaches to the straight line when the rate of increase of the load approaches the zero.*

The elasticity of the muscle determined from the beginning of the extension curve is 1·08. The determination of this quantity is approximately possible, because in the beginning, when the load is very small, the after-elongation vanishes against the elastic elongation. It is also possible to determine the elasticity of the muscle from the end of the tracings, which is strictly rectilineal. The elasticity determined from that part of the curve amounts to 1·06, being the same as at the beginning, within the limits of experimental error. I conclude from this agreement *that the elasticity of the muscle is constant and that the deviation from the straight line in the beginning of the curve is caused by the after-phenomenon.* The sixth row of Table XIV gives the plasticity of the muscle, calculated upon this base. We see that under the given régime the plasticity of the muscle is very small in comparison with the elasticity. In fig. 11 I plotted the plasticity against the time. The figure shows that at first the plasticity quickly increases, reaches a maximum, and then decreases.[1]

Experiment 13 (Table XV) is of the same kind. The mean increase of the load, however, was larger, viz., 27 mg. per second. The deviation from the mean was not so considerable as in Experiment 12. After three-quarters of an hour the maximum charge was reached, and from that moment on the load decreased. The rate of decrease of the load was fairly constant in the beginning, but slowly diminished towards the end of the experiment. Eighty-six minutes after the commencement of the experiment the muscle was again under its initial charge. From that moment onwards I observed the after-shortening of the muscle for about thirty minutes, and found that the relation expressed by the formula $\left[\frac{\Delta L}{\Delta \tau} \times \tau = \text{const.}\right]$ is not satisfied. In this case the value of the constant first increases and then probably decreases. I found the same result in other experiments, so *that the relation* $\left[\frac{\Delta L}{\Delta \tau} \times \tau = \text{const.}\right]$ *only holds good when the time required for the loading and the unloading of the muscle is small in comparison with the time necessary for the whole*

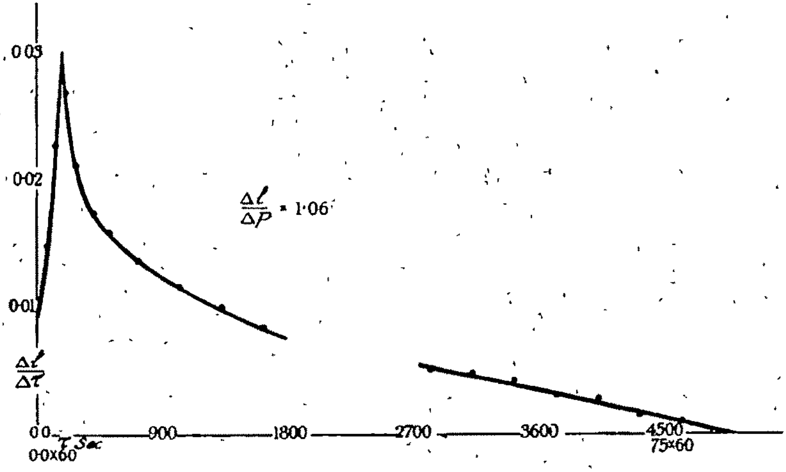

FIG. 11.—Graphic representation of the result of Experiment 12. The plasticity as a function of the time.

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[1] The gap in the curve is caused by a slight discontinuity in the original tracing.

cyclical variation; in other words, when the charging and discharging of the muscle takes place fairly quickly.

The elasticity determined from the beginning and from the end of the extension curve amounts to 1·25. Fig. 12 represents graphically the plasticity of the muscle. This curve resembles closely the curve of fig. 11, but from the scale upon which the curve is drawn it follows that the plasticity is about double in this case. The unloading curve is in many respects similar to the extension curve. The elasticity, determined from the beginning of the unloading curve, amounts to but 0·18. This fact proves that the muscle has undergone a plastic deformation. The plasticity curve (fig. 13), derived from the falling-load curve, resembles the plasticity curve of the rising-load tracing, but inverse. The arrow at the abscissa indicates the moment when the muscle is again under its initial charge. From that moment on the plasticity falls and then slowly slopes towards zero. Half an hour after the accomplishment of the cycle there is still an elongation of thirty-seven units.

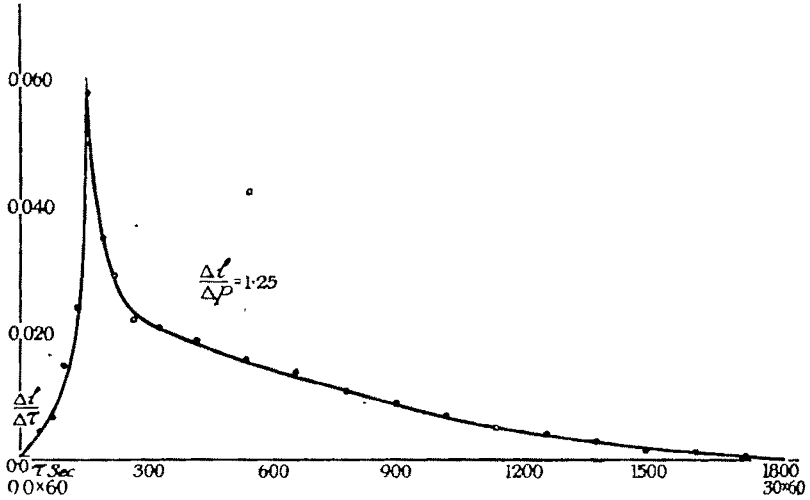


FIG. 12.—Graphic representation of the result of Experiment 13. The plasticity as a function of the time during the loading of the muscle.

In the three last experiments the increase of the load took place quickly and uniformly, the rate of increase of the charge varying from about 3 to 0·6 grm. per second in the successive experiments. The unloading happened abruptly and in a somewhat irregular manner, so that the period immediately following the unloading was not suitable for measurement. The zero of time necessary for the determination of the constant of plasticity is therefore a little uncertain, and also the length of the muscle immediately after the unloading.

Experiment 14, *December* 20, 1904.—The muscle was cut out at 10·05 a.m., and suspended in the moist chamber at 10·12 a.m. under an initial charge of 10·75 grm. Some minutes later the experiment commenced; it consisted of eleven cyclical variations of the load with equal intervals of about ten minutes. The rate of increase of the load was approximately 3 grm. per second, and the charging of the muscle took nearly twenty-five seconds. The muscle was left for about five seconds under the influence of the maximum charge and then abruptly unloaded.

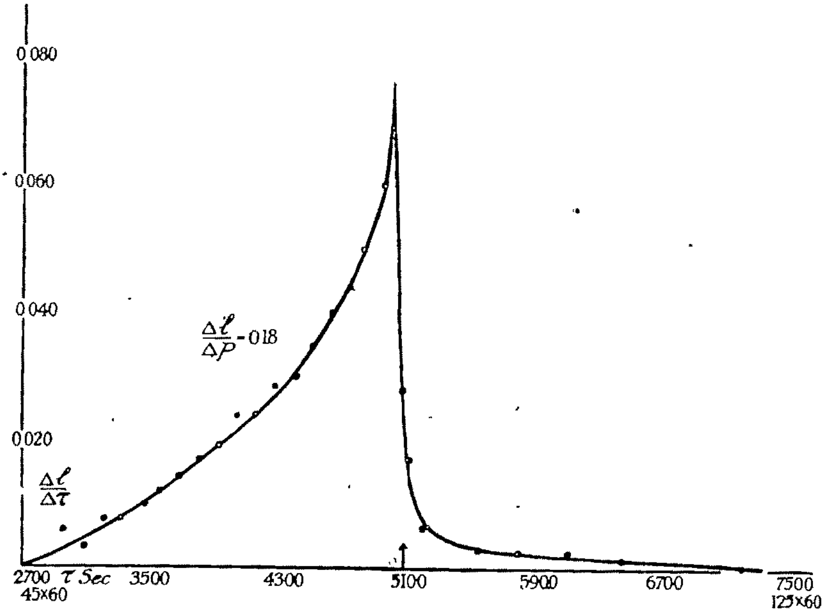


Fig. 13.—Graphic representation of the result of Experiment 13. The plasticity as a function of the time during the unloading of the muscle.

At the moment when the muscle was under full and constant charge we may determine the amount of the after-lengthening per second, because under these conditions the increase in length of the muscle is only due to the after-phenomenon. At the moment just before the maximum charge was attained we may determine the increment in length of the muscle provoked by the joint effect of the after-phenomenon and the increment of the charge. The difference between these two quantities gives approximately the pure elastic strain of the muscle at the end of the extension curve. The elasticity of the muscle may also be determined from the beginning of the extension curve, when the increment of the load is small, and the after-lengthening therefore vanishes against the elastic elongation. In these experiments, too, the elasticity of the muscle, determined from the beginning and from the end of the tracing, is always the same within the limits of experimental error. We conclude, therefore, *that in these experiments also, where the extension curve deviates considerably from the straight line, the deviation is caused by the after-phenomenon.* The determination of the plasticity is based on this conclusion.

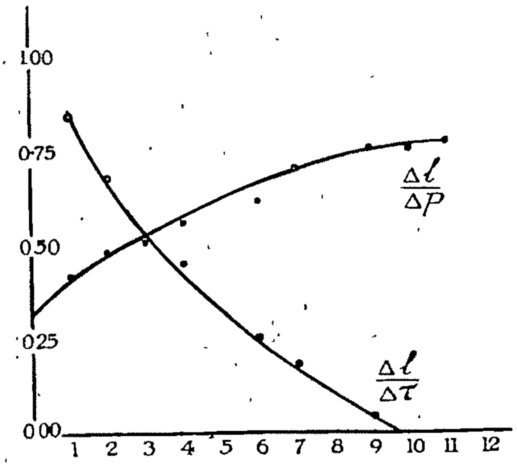


FIG. 14.—Graphic representation of the result of Experiment 14. The curve at the end of which the notation $\frac{\Delta L}{\Delta P}$ is placed represents the elasticity of the muscle in the successive cycles. The curve at the end of which the notation $\frac{\Delta L}{\Delta \tau}$ is placed represents the plasticity of the muscle at the moment of the maximum charge.

Tables XVI, XVII, and XVIII contain the measurements of the first, the sixth, and the tenth curves. Of the other curves of this series I give only the numerical results combined in Table XIX. The first row of this table contains the time necessary for the loading of the muscle in seconds; the second row the rate of increase of the load in grammes per second; the third row the elasticity of the muscle; and the fourth row the constant of plasticity during the period of after-shortening. It is evident from this table that the régime was fairly the same in the eleven cycles. The variations in the elasticity and in the plasticity of the muscle in the course of the experiment are therefore only due to the repetition of the cyclical variation of the load. In figs. 14 and 15, the results are plotted. The numbers along the abscissa denote the successive cycles. These numbers are placed equidistant because the cycles succeeded each other with equal intervals of time. The first curve (fig. 14), at the end of which the notation $\frac{\Delta L}{\Delta P}$ is placed, represents the variation of the elasticity of the muscle in the successive cycles. We conclude from this graph *that the elasticity of the muscle is increased by repeating the cyclical variation of the load.* It is evident from the form of the curve that this quantity approaches an upper limit. A rough extrapolation makes it probable that this limit lies in the neighbourhood of 1, and that this limit would be attained after about 25 to 30 cycles. The second curve, marked $\left[\frac{\Delta L}{\Delta \tau}\right]$, represents the plasticity of the muscle exactly at the moment when the maximum charge is arrived at. The meaning of this curve is to show that the area over which the after-elongation extends narrows successively. Fig. 15 represents the constant of plasticity during each interval succeeding a cyclical variation of the load. The points representing this quantity are all grouped along a straight line, as already found (Experiments 8 and 9). By extrapolating the

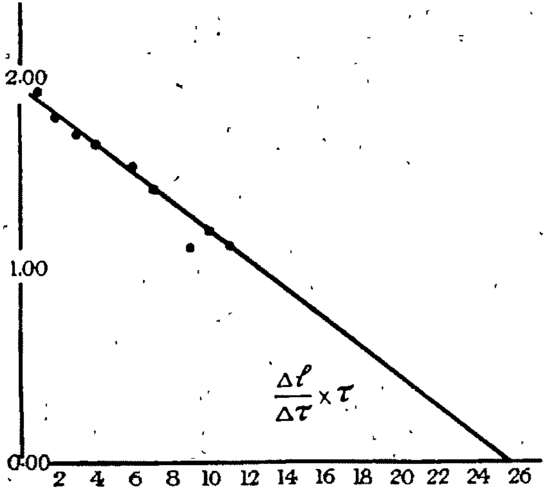


FIG. 15.—Graphic representation of the result of Experiment 14. The constant of plasticity during each interval succeeding a cyclical variation of the load. The numbers along the abscissa denote the successive cycles.

line it would cut the abscissa in the neighbourhood of Number 26. This means that after 26 cycles the muscle immediately after the unloading would reach its initial length. When this is the case, the after-phenomenon is eliminated. This extrapolation tends, therefore, to the same conclusion as the extrapolation of the curve of fig. 14, viz., *that by repeating a rapid cyclical variation of the load the elastic after-phenomenon is reduced in extent and in magnitude, and that probably* 25 *to* 30 *cycles would suffice to eliminate the after-phenomenon.* At that moment the elasticity is maximum and the muscle reduced to a purely elastic body. Brodie [13] arrived in a somewhat different way at a similar result. Fig. 16 is the reproduction of the plasticity, deduced from the first, sixth and tenth extension curves. The tracings exhibit the same feature as those of figs. 11 and 12. A comparison, however, of the scales shows that the plasticity is in this case about 500 times larger than in Experiment 11, and that the time needed for the full development of the after-phenomenon is much smaller.

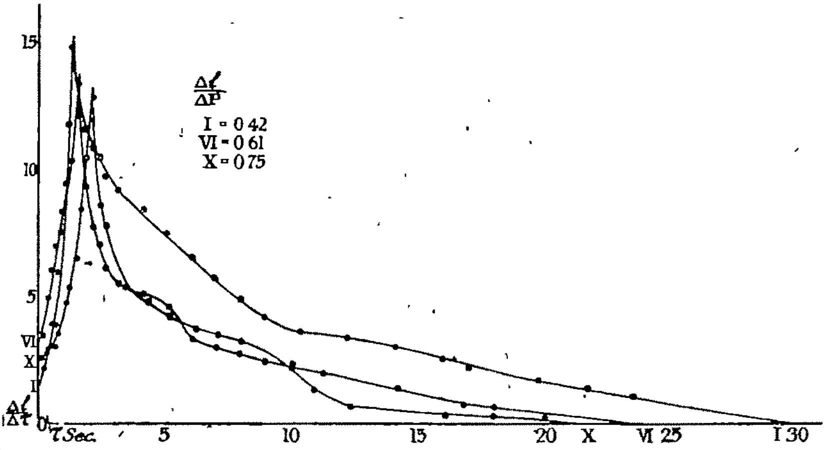


Fig. 16.—Graphic representation of the results of Experiment 14. The plasticity as a function of the time during the loading of the muscle—first, sixth and tenth extension curves.

Experiment 15 is of the same nature as Experiment 14, but the rate of increase of the load is somewhat smaller. Table XX contains the chief results, and figs. 17 and 18, are the graphs plotted in the same way as figs. 14 and 15. The extrapolation of the curve marked $\left[\frac{\Delta L}{\Delta P}\right]$ of fig. 17 tends to the conclusion that the upper limit to which the elasticity approaches lies in the neighbourhood of 0·75, and would

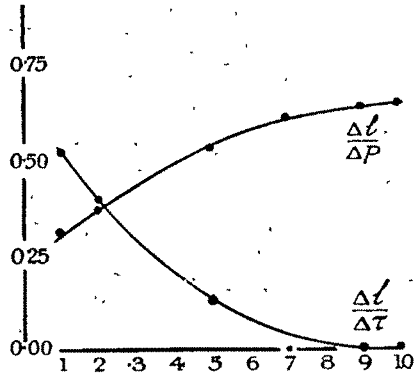


FIG. 17.—Graphic representation of the result of Experiment 15. The curve at the end of which the notation $\frac{\Delta L}{\Delta P}$ is placed represents the elasticity of the muscle in the successive cycles. The curve at the end of which the notation $\frac{\Delta L}{\Delta \tau}$ is placed represents the plasticity of the muscle at the moment of the maximum charge.

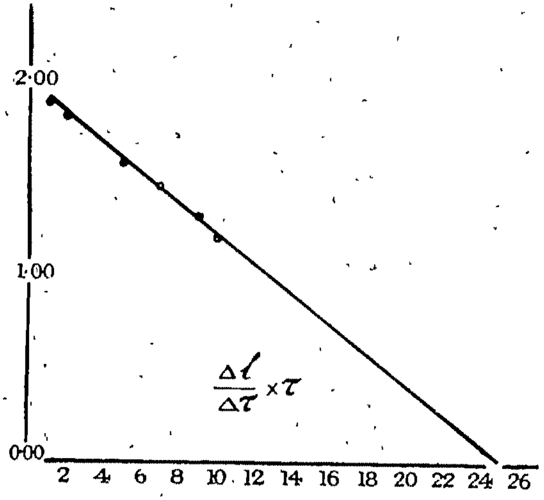


FIG. 18.—Graphic representation of the result of Experiment 15. The constant of plasticity during each interval succeeding a cyclical variation of the load. The numbers along the abscissa denote the successive cycles.

be reached after 20 to 30 cycles; the extrapolation of the straight line of fig. 18 tends to the same conclusion.

Experiment 16 is the last of this group. In this case the rate of increase of the load is still smaller, about 0·65 grm. per second. In Table XXI the results are combined; figs. 19 and 20 are the graphs. Extrapolation of the $\left[\frac{\Delta L}{\Delta P}\right]$ curve proves that the upper limit of the elasticity lies probably between 0·75 and 1, and would be reached after 20 to 25 cycles. The extrapolation of the straight line of fig. 20 is in harmony with this. Fig. 21 is the reproduction of the plasticity curves, deduced from the first, third and eighth extension curves; these curves resemble closely those of fig. 16, but the scales are different.

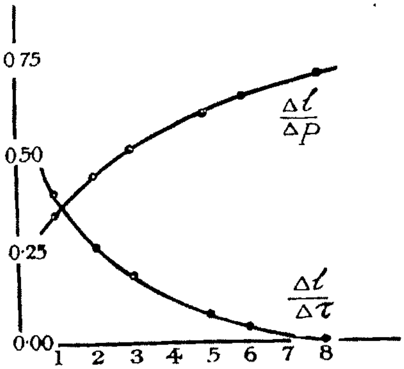


FIG. 19.—Graphic representation of the result of Experiment 16. The curve at the end of which the notation $\frac{\Delta L}{\Delta P}$ is placed represents the elasticity of the muscle in the successive cycles. The curve at the end of which the notation $\frac{\Delta L}{\Delta \tau}$ is placed represents the plasticity of the muscle at the moment of the maximum charge.

It is evident from the comparison of the plasticity curves reproduced in figs. 11, 12, 16 and 21 that these curves resemble each other in their general features. The chief difference is in the scales upon which these tracings are drawn. A comparison of these scales proves that the plasticity increases when the rate of increase of the load grows larger. It is obvious that this result is inherent to the method I employed to measure the plasticity, and does not lie in the properties of the muscle. We conclude, therefore, *that the atonic muscle at the moment it is cut out of the body is a very plastic body, but that physical* *contrivances of different kinds, e.g., continued pull, cyclica of the load, vibration, &c., tend to eliminate the plasticity an the atonic muscle to a purely elastic body.*

Finally, I will try to formulate in clinical terms the res part of my researches. The muscle, as soon as it becon gradually increases in length. This increase is caused by the pull to which it is exposed by the tonus of its antagoni stretching to which it is subjected during movement, by the to which it is subjected during changes in posture of the body final length to which the muscle tends is probably identica

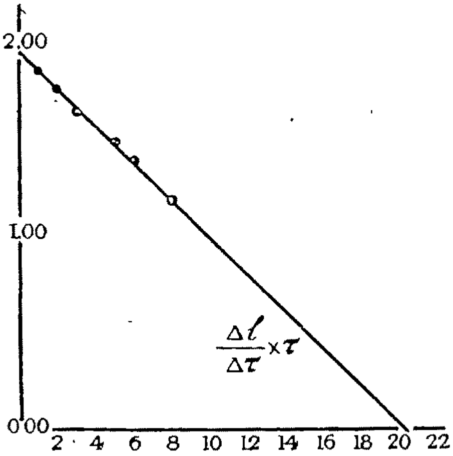


FIG. 20.—Graphic representation of the result of Experiment 16. The constant of plasticity during each interva succeeding a cyclical variation of the load. The numbers along the abscissa denote the successive cycles.

post-mortem length. In consequence of its increased length hangs loose and flabby between its insertions; in displacing as a whole, to and fro across its length no active resista Pressed between thumb and index the muscle feels solid, lik has not the softness of the tonic muscle.

(*B*) *The Temperature Variable.*

The influence of the temperature upon the length of muscle has often been studied, and after the classic expe Brodie and Richardson [14] and the more recent work of J and others, my own researches are only of subordinate intere for these experiments the M. gastrocnemius of the frog. The muscle was cut out of the body and suspended in a small moist chamber. The moist chamber was surrounded by a closed mantle, in which two streams, one of cold water and one of hot water, could circulate. In this way the temperature of the moist chamber was varied. A thermometer was placed in the moist chamber, quite near the muscle, so that the fluid which kept the muscle wet formed a thin continued film round the muscle and the thermometer. The muscle was fastened to a length recorder, which registered the length of the muscle upon a rotating drum, upon which the time was also recorded. Every minute I noted the temperature of the thermometer in contact with the muscle. The curves recorded in this way are by intermediation of the time length-temperature diagrams. This method is rather rough, and the construction of the thermostat too simple to give very exact results, but for my purpose it was sufficient.

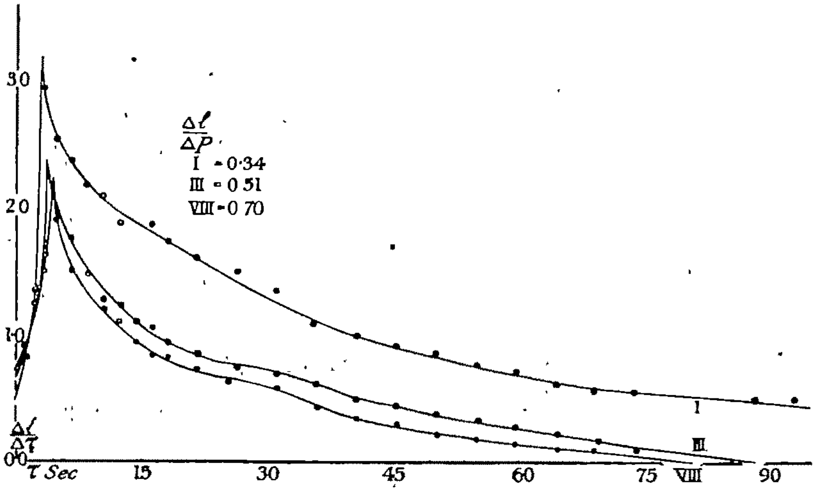


FIG. 21.—Graphic representation of the results of Experiment 16. The plasticity as a function of the time during the loading of the muscle—first, third and eighth extension curve.

The units adopted in this part of the researches are: 1° C. as the unit of temperature [T]; one minute as the unit of time [τ] and 1 mm. as the unit of length [L, l]. The enlargement by the length recorder amounted to 5·55, so that the real unit of length is $\frac{1}{5\cdot 55}$ mm. The increase in length of the muscle is considered as a positive variation of L [+], the decrease of the length as a negative one [—].

The aim of these experiments was to establish a condition of uniformly varying temperature. The tables show that the condition of uniformity is not fulfilled; the curves, however, which I obtained are notwithstanding fairly regular. This result is only caused by the fact that the influence of the rate of variation of the temperature is not very marked upon the form of the length-temperature diagram. On the other hand, there is a compensation factor, viz., that the muscle, as a whole, follows but slowly the variation of the temperature of the ambient medium.

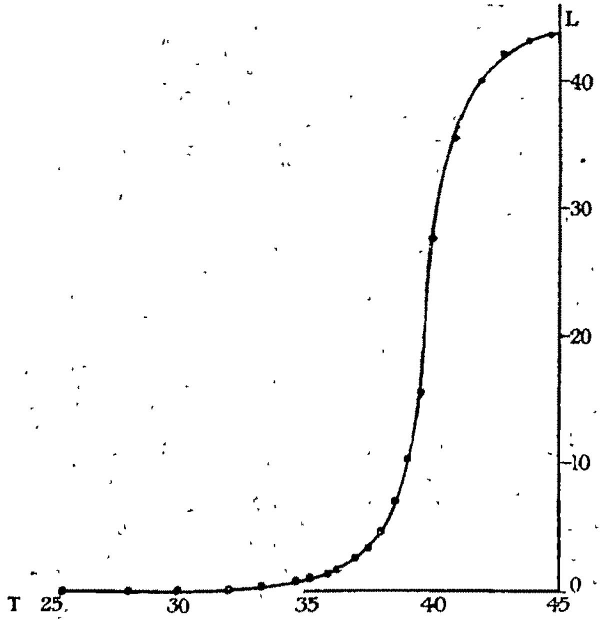
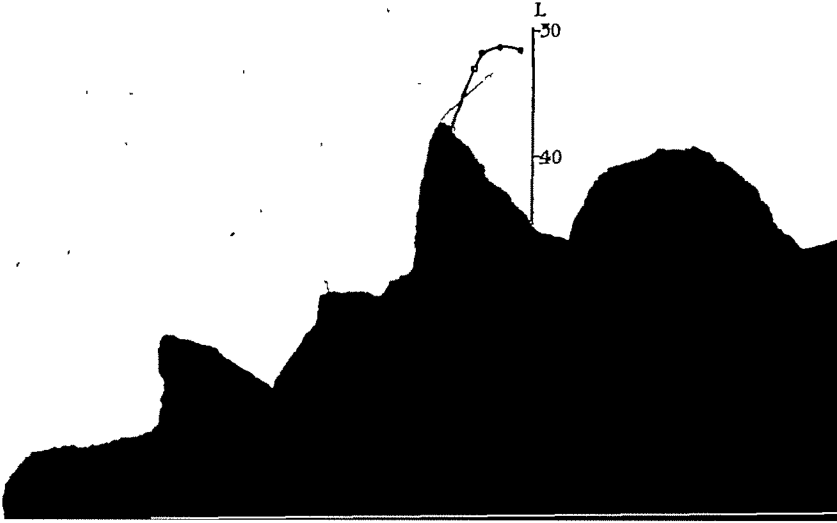


FIG. 22.—Length-temperature diagram of Experiment 1.

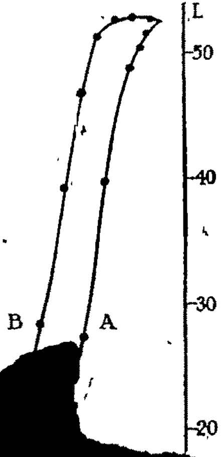
The muscle shortens in consequence of the increase of the temperature, until a first maximum is reached. This maximum lies in my experiments in the neighbourhood of 45° C. Beyond this first maximum I have not extended my experiments.

Experiment 1, *December* 18, 1906.—The frog was killed at 9.45 a.m.; at 10 a.m. the muscle was cut out, suspended in the moist chamber and charged with 14·9 grm. The experiment began at 10.17 a.m., but previously I performed ten cyclical variations with the load, partly to free the muscle from the after-phenomenon. It follows from Table XXII, which contains the result of the experiment, that by these cyclical variations of the load the muscle is brought to a state of rest. More strictly speaking I ought to say *that the muscle has not come to a state of rest, but oscillates between very narrow and fixed limits.* The third row of Table XXII contains under $\left[\frac{\Delta T}{\Delta \tau}\right]$ the rate of the variations of the temperature, i.e., the increase or decrease of the temperature per minute. The mean value of $\left[\frac{\Delta T}{\Delta \tau}\right]$ during the interval 27° C. to 45° C. is about 0·3° C. per minute. The thermic shortening of the



temperature diagram. In this case the curve is more flat and the S shape not quite so pronounced as in fig. 22. This result leads to the supposition that the rate of increase of the temperature might influence the form of the length-temperature diagram.

I tried to verify this supposition in the following way: First I took the left M. gastrocnemius of the frog and warmed the muscle rather slowly, then I took the right gastrocnemius and raised the temperature



curves of the Experiments 1 and 2. We conclude, therefore, *that the rate of increase of the temperature influences the form of the length-temperature diagram.* One might object to this experiment on the ground that the two gastrocnemii of the same frog, though originally equal, are no longer alike at the moment of the experiment, because the experiment with the second gastrocnemius always began one to two hours after the experiment with the first muscle. The experiment shows, however, that in both cases the same final length was attained. This fact is in favour of the view that the difference between the A and B curves is chiefly caused by the difference in the rate of increase of the temperature.

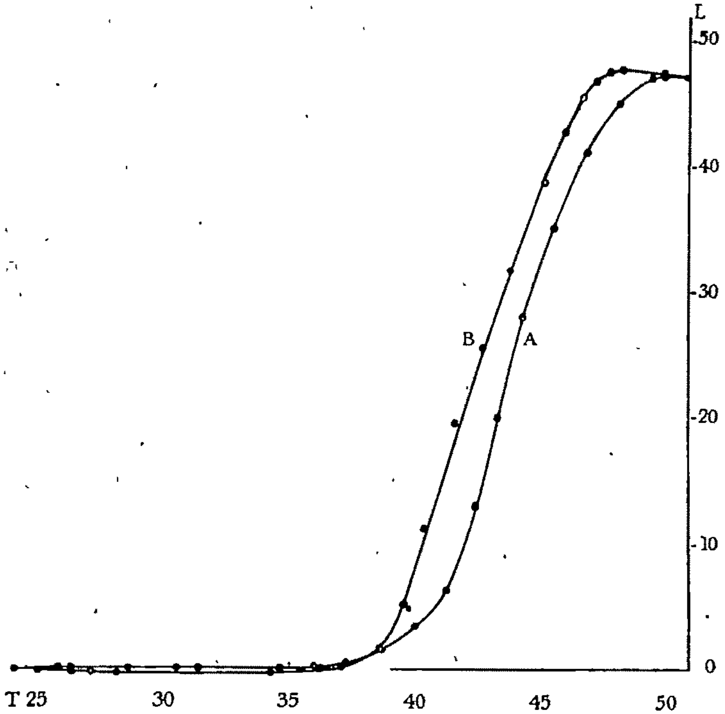


FIG. 25.—Curve A, length-temperature diagram of Experiment 5. Curve B, length-temperature diagram of Experiment 6.

The result at which we arrived may also be formulated in a somewhat different manner. We may conclude *that only the final state is fixed by the temperature of the muscle, but that the length of the muscle, at a moment intermediate between the beginning and the end of the shortening, is not merely determined by the temperature, but also by* *the way in which this temperature is reached.* Formulated in this manner, we can prove the results of the foregoing experiments by cyclical variations of the temperature.

Experiment 7, *December* 1, 1906, is composed of three non-closed thermal cycles. The measurements of the original tracing are combined in Table XXVIII, and the length-temperature diagram is reproduced in fig. 26. First the temperature is raised from 12·4° C. to 32·5° C., with a mean increment of 0·48° C. per minute. Then the temperature falls from 32·5° C. to 21·5° C., with a mean rate of decrease of 1·37° C. The rise of the temperature is accompanied by a shortening of the muscle [rising curve], the fall of the temperature is followed by a lengthening of the muscle [falling curve]. In the first cycle the falling curve lies above the rising curve; this result is a general one, the falling curve always lying above the preceding rising curve, and we conclude, therefore, *that the increase of the temperature produces a shortening of the muscle, and that this shortening is partly permanent.*

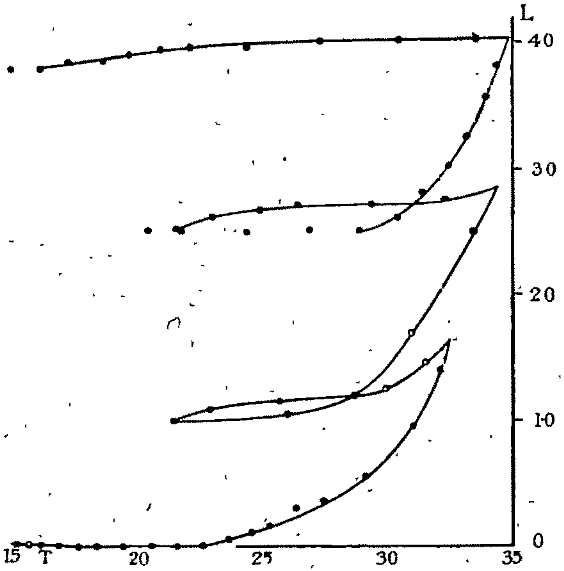


FIG. 26.—Length-temperature diagram of Experiment 7.

The curve corresponding to the falling temperature is also S-shaped, but as the temperature at which the reversion from rising to falling temperature takes place becomes higher, the falling curve loses more and more its S-shaped form; at last the falling curve is nearly a horizontal line. This fact is illustrated by Experiment 8, where the points of the reversion of the temperature lie higher than in Experiment 7. We conclude, therefore, *that the rise of the temperature produces a gradually increasing deformation of the muscle.*

The second thermal cycle of Experiment 7 is composed by an interval of increasing temperature from 21·5° C. to 33·5° C., with a mean increment of 1·38° C. per minute. This interval of rising temperature is followed by an interval of decreasing temperature from 33·5° C. to 20° C., with a mean decrement of 1·86° C. The rising curve of the second cycle cuts the falling curve of the preceding cycle and forms a loop.

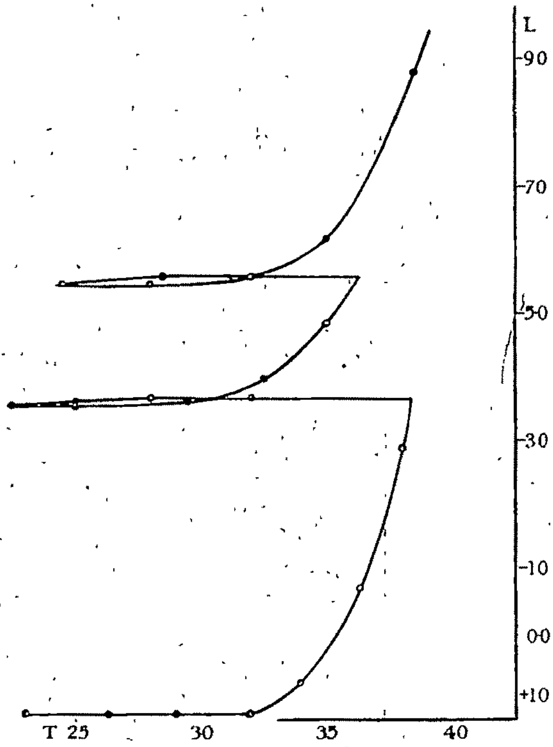


FIG. 27.—Length-temperature diagram of Experiment 8.

The third thermal cycle begins with an interval of increasing temperature, from 20·5° C. to 34·4° C., followed by an interval of decreasing temperature, from 34·4° C. to 15° C. The mean value of the variation of the temperature during the rising interval is 1·40° C., during the falling interval, 1·76° C. per minute. The second loop forms a peculiarity. This loop is incomplete. As soon as a temperature of 21·7° C. is reached, the muscle ceases to lengthen, notwithstanding the temperature falling further to 20·5° C. At that moment the third thermal cycle begins, and the temperature again increases. The muscle, however, remains at constant length until the temperature reaches 29° C.; then a new period of shortening sets in. A part of the second loop, therefore, cannot be realized, in consequence of the permanent thermal deformation of the muscle.

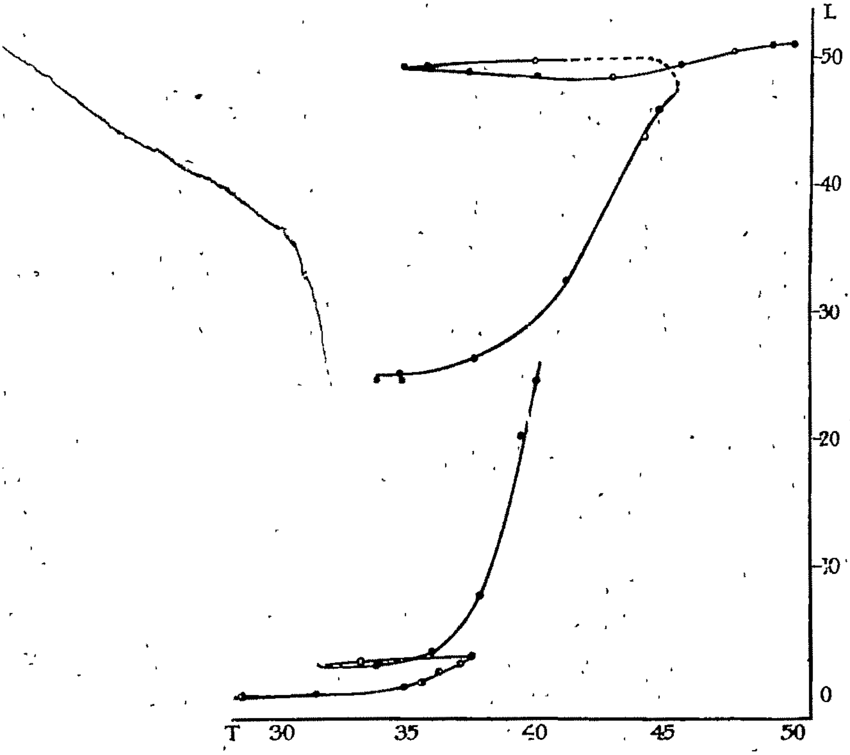


Fig. 28.—Length-temperature diagram of Experiment 9.

Experiment 8 is of the same nature as Experiment 7. The points of reversion from rising to falling temperature, however, lie higher. Table XXIX contains the measurements, and fig. 27 is the reproduction of the length-temperature diagram of this experiment.

The last experiment of this kind is No. 9. It is composed by three thermal cycles. The second loop could not be realized, and the third loop has a somewhat anomalous appearance. This is caused by the well-known fact that in the neighbourhood of the first maximum, increasing temperature may be accompanied by a lengthening of the muscle. Table XXX gives the measurements, fig. 28 is the pertaining length-temperature diagram, and fig. 29 the original tracing. We conclude from these experiments *that the length of the atonic muscle is not entirely determined by its temperature, and inversely that a fixed temperature does not agree with a determined length of the muscle.* In other words, the length of the muscle is greatly determined by its physical and chemical deformation, and the amount of this deformation at a certain moment depends not only upon the actual temperature of the muscle, but also upon the way by which this temperature is reached.

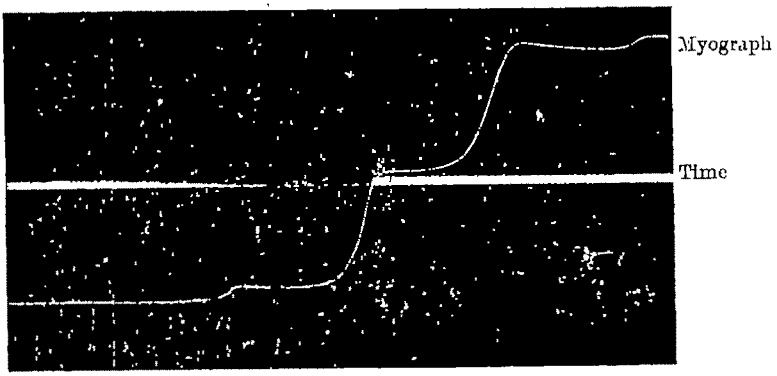


FIG. 29.—Reproduction of the myograph of Experiment 9. The enlargement is about 3·7 times.

Finally, I combined in a table the quantities, which are of interest for the knowledge of the process of thermal shortening. Ten of my experiments were suitable for this purpose. For each of these quantities I determined the arithmetical mean of these ten observations. In this way I found that the thermal shortening of the frog's gastrocnemius[1] begins between 28·5° C. and 29·5° C., that the first maximum of the shortening is reached in the neighbourhood of 46° C. and that the total amount of the shortening at that moment attains to 26 per cent.

The question of the thermal coefficient of the muscle during the interval preceding the thermal shortening is much discussed. If we define the term "thermal coefficient" as the variation in length in millimetres for 1° C. per centimetre length of the muscle I find :—

---

[1] I used for these experiments *Rana esculenta,* and the experiments were performed in winter.

$\frac{\Delta L}{\Delta T} \times \frac{1}{L} \overset{=}{<} 74 \times 10^{-5}$ for the interval 12° C. to 27° C.

[Experiment 1, dated December 18, 1906.]

,, $\overset{=}{<} 60 \times 10^{-5}$ for the interval 12·4° C. to 22·7° C.

[Experiment 7, dated December 1, 1906.]

,, $\overset{=}{<} 84 \times 10^{-5}$ for the interval 9·8° C.—25·3° C.

[Experiment 10, dated December 12, 1906.]

These observations prove that the thermal coefficient of the muscle is extremely small. In some experiments I found the phenomenon described by Brodie and Richardson, viz., that the muscle during the interval of temperature below the temperature at which the thermal deformation is manifest, may lengthen in consequence of a rise in temperature. I observed this phenomenon in some cases in the neighbourhood of 20° C. In these cases, however, it was the after-lengthening, which was again activated by the rise of the temperature, for I never saw the phenomenon when I previously eliminated the after-phenomenon by cyclical variations of the load. Moreover, the magnitude of the lengthening agreed with the lengthening caused by the after-phenomenon and was by no means of the order of magnitude of the thermal coefficient. We conclude, therefore, *that the thermal coefficient of the atonic muscle is extremely small within the limits of temperature which are of physiolcgical interest, or small variations of the temperature round the physiological norm do not sensibly influence the length of the atonic muscle.*

CHAPTER II.—THE TONIC MUSCLE.

My researches on the atonic muscle have proved that the muscle possesses two fundamental properties, viz., elasticity and plasticity. The aim of this second part of my investigation is to trace the rôle which these two properties play in the function of the tonic muscle. To that purpose, I make use of my experimental material of the years 1899, 1900 and 1901. A part of this material was published in *Archiv für Physiologie*, Jahrgang. 1901 and 1902, but the analysis given there was incomplete. It was the study of the atonic muscle which enabled me to analyse more completely the function of the tonic muscle. The morphological researches of the latter years also contributed to this result, since it was demonstrated by Boeke that striped muscle possesses a double motor innervation. He showed that the striped muscle is not only innervated by the well-known axone of the motor cell of the anterior horn, but also by a motor sympathetic fibre. This fact makes the image of the simple proprioceptive reflex arc more complicated than it was before. For the sake of brevity and clearness I propose to give first a scheme (fig. 30) of the proprioceptive reflex arc, in which are incorporated the joint results of the morphological researches of Boeke [2], [3], [4], [5], [6] and of my own physiological investigations.

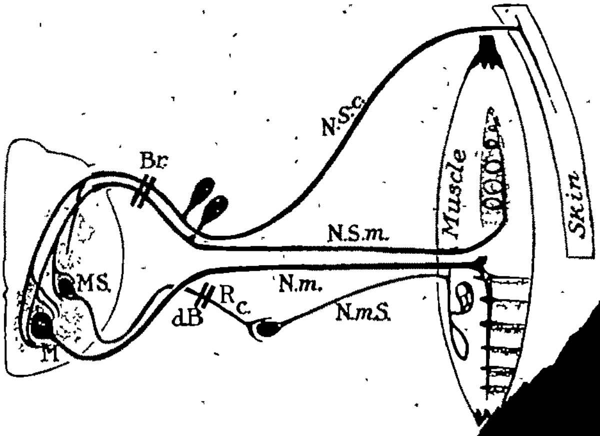


FIG. 30.—Scheme of the proprioceptive re[illegible] place at which Brondgeest interrupted th[illegible] reflex arc; d.B, place where de Boer i[illegible] prioceptive reflex arc; M, motor cell[illegible] M.S., sympathetic motor cell; N.m[illegible] along the edges of the anisotropic d[illegible] efferent [illegible] S.c., af[illegible] [illegible]

terminate in the motor end-plate of the muscle-fibre, but according to the researches of Boeke does not really end there. Fine filaments leave the net of neurofibrils situated in the end-plate and run parallel to the striped muscle-fibre. They in that way give off finest filaments at a right angle, which extend along the edge of the anisotropic disc, where they seem to end. It appears, therefore, that a very close connexion exists between the axone of the motor cell of the anterior horn and the striped apparatus of the muscle.

The other of the two motor cells is a sympathetic cell. The axone of this cell leaves the cord along the anterior root and passes over into the sympathetic chain by way of the grey ramus communicans. When this sympathetic fibre reaches the muscle, it forms a non-myelinated sympathetic plexus, from which fine filaments enter the muscle-fibre. These filaments end in fine nets of neurofibrils, which are embedded in [illegible]all sarcoplasmatic end-plate. In general the sympathetic cell does [illegible] in the same level as the motor cell of the anterior horn. It [illegible]m experiments of de Boer [7], [8], [9], and of Keere, [illegible]and Naito [26] that the sympathetic cells are situated in [illegible]art of the cord, and that they belong to the thoracic [illegible]m.

[illegible]cal researches of Boeke lead, therefore, to the con- [illegible] *of the striped muscle, viz., a sarcoplasmatic mass,* [illegible]*hetic fibre analogous to a smooth muscle, in* [illegible] *apparatus. This apparatus is in close* [illegible]*rmination of the axone of the motor cell* [illegible]n of the duality of the striped muscle [illegible]tazzi [11], [12], [illegible] [35], [36] [illegible] blood-vessels were spared, so that the muscle remained in quite normal relation with its proprioceptive reflex arc. A light recipient was fixed to the tendon by means of a hook. A stream of mercury flowed in the recipient with constant velocity, so that the charge of the muscle increased uniformly with the time. As soon as the charge reached a certain maximum the inflow was stopped, and then after some seconds the muscle was unloaded abruptly. This experiment was repeated several times with approximately equal intervals. The experimental condition established in that way is therefore one of successive cyclical variations of the load. The variation of the length of the muscle under the influence of the varying load was recorded upon a rotating drum upon which the time was also noted. The curves obtained in this manner are extension curves of the tonic muscle.[1]

The experiment of making extension curves of the tonic muscle by means of a uniformly increasing weight is not as simple as it seems to be, for in the first place the weight acts as a purely physical factor, as a load that extends the muscle, but in the second place it acts as a physiological agent. The stretching of the muscle by means of the increasing weight forms an adequate stimulus for the proprioceptors of the muscle to which it answers with a slow contraction. *The extension curve of the tonic muscle is therefore an extension curve, not of a resting muscle but of a contracting one.*

The fact that the tonic muscle, when stretched by an increasing weight, really contracts is demonstrated by the experiments of Buytendijk [15] and Samkow [39]. Buytendijk observed the action current of the tonic M. vastocrureus of the cat, and found that the intensity of this current augments, when the load attached on the tendon increases, till a certain maximum is attained.

The units used in this part of the research are: 1 second as the unit of time [τ]; 1 cm. as the unit of length [L, l]; 1 grm. as the unit of weight [P, p].

The analysis of the extension curve of the tonic muscle is based upon the following considerations. The extension curve is by intermediation of the time a length-load diagram, i.e., the graphic record of the relation between the length of the tonic muscle and the load attached on its tendon. It is therefore possible to determine directly from those tracings the increase in length of the muscle per gramme increase of the load. This quantity I have defined, in my work published in *Engelmann's Archiv*, as the *tonus* of the muscle, in accordance with the earlier observers. The study of the atonic muscle has proved, however, that this quantity comprises two different elements, viz., an elastic elongation and an after-elongation, or, in other words, *that the tonus is the sum of an elasticity and of a plasticity.* The quantity defined as tonus has the advantage of being a directly measurable quantity, but the disadvantage that it does not allow of a more complete analysis of the properties of the tonic muscle. For the moment I will represent the tonus of the muscle at rest by the letter "a."

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[1] A full account of the technique is given in *Engelmann's Archiv*, Jahrgang 1901, S. 106.

The stretching of the tonic muscle by means of a uniformly increasing weight stimulates the proprioceptors and causes the muscle to contract. This contraction produces a variation of the length of the muscle, the value of which per unit increment of the load may be represented by $[\frac{\Delta l}{\Delta P}]$. Hence the tonus of the muscle under the conditions of the experiment ought to be represented by $[a - \frac{\Delta l}{\Delta P}]$. We have therefore $[a - \frac{\Delta l}{\Delta P}]$ = muscular tonus = the increase in length of the tonic muscle per gramme increase of the load. My earlier observations have proved that there exists a connexion between the stretching weight and the tonus, viz., *when the weight increases in geometrical progression the tonus decreases in arithmetical progression.*

Expressed in mathematical symbols we find—

$$e^{C_2 \left(a - \frac{\Delta l}{\Delta P}\right)} = C_1P \text{............IA; or,}$$

$$C_2 \left(a - \frac{\Delta l}{\Delta P}\right) = \text{lgn. } C_1P \text{ ..IB.}$$

in which formulæ—

$\left(a - \frac{\Delta l}{\Delta P}\right)$ = tonus,

P = the stretching weight,

C_1 and C_2 = two constants.

The stretching weight P functionates as a stimulus for the proprioceptors, so that we may also say that $[C_1P]$ represents the value of the stimulus, and $[C_2(a - \frac{\Delta l}{\Delta P})]$ the corresponding value of the tonus. Formula I states, therefore, *that there exists a logarithmic relation between the stimulus and its effect.*

The physiological interpretation of the result of my experiment makes it necessary to introduce the threshold value Δ into the formula, whereby this formula assumes the form:—

$$C_2 \left(a - \frac{\Delta l}{\Delta P}\right) = \text{lgn. } C_1(P - \Delta) \text{..............IC,}$$

$[P-\Delta]$ being the excess of the stimulus above the threshold value. Formula IC may be integrated, if we substitute an integration for a summation. We find as the result of the integration—

$$l = a\,P - \frac{1}{C_1C_2}[C_1(P-\Delta)\{\,\text{lgn.}\ C_1(P-\Delta)-1\,\}]\ \text{II};$$

this formula expresses the relation between the increase in length of the tonic muscle $[l]$ and the increase of the weight $[P]$.

Formula II states more particularly that the increase in length of the tonic muscle may be considered as the difference of two quantities, viz.—

$$[a\,P] \text{ and } \frac{1}{C_1C_2}[C_1(P-\Delta)\{\,\text{lgn.}\ C_1(P-\Delta)-1\,\}].$$

The first of these quantities, according to the definition given of a, is the total increment in length of the muscle caused by the increase of the weight P, if the muscle did not contract. The second quantity, in agreement with the genesis of the integrated formula, represents the shortening of the muscle in consequence of the slow contraction, caused by the stimulation of its proprioceptors. The increase in length of the tonic muscle under the conditions of the experiment is therefore the sum of a purely physical elongation, and a shortening caused by a physiological process.

This result conforms with the experimental data of the first part of this research, for if the proprioceptive reflex arc is broken, the physiological effect vanishes, and formula II is reduced to $l = a\,P$, i.e., the increment in length of the atonic muscle is the sum of the elastic elongation and of the elastic after-elongation. For the case the after-phenomenon is eliminated, $l' = a\,P$ represents the law of Hooke. In the third part of this investigation I will show that the formula

$$\frac{1}{C_1C_2}[C_1(P-\Delta)\{\,\text{lgn.}\ C_1(P-\Delta)-1\,\}]$$

may represent the relation between the stimulus and the lift of a muscle contracting under isotonic conditions.

First I will prove that the experimental results may be represented by the analytical expression IC. The experiments used to that purpose were for the greater part already published in *Engelmann's Archiv*, 1901. The numbers placed in brackets behind the numbers of the experiments refer, therefore, to the Tables and to the reproductions of the original tracings in that publication. I repeated, however, the measurement of the length-load diagrams and corrected thereby little faults in my earlier measurements. I moreover extended my measurements to parts of the curve which are now of interest for the more complete knowledge of the problem, but which at that time were neglected.

Experiment 1 [*Table II*], *September* 15, 1899. — The frog used for this experiment was intact, but by wrapping up the whole animal in wet cotton-wool unintended stimuli were fairly well eliminated. The successive extension curves of the tonic M. gastrocnemius were made with approximately equal intervals of some minutes. The curves thus obtained resemble each other closely, and the course of the whole experiment was not troubled in any way. In some other experiments the cord was previously cut through, but, at least in the frog, there was no appreciable difference in the tracings in either case, except a slight depression of the tonus by shock, shortly after the section of the cord. Table XXXI gives the measurements of the fifth extension curve. The first row of this table contains the time [τ], the second row the charge [P], the third row the increment in length [L] of the tonic muscle, caused by the weight [P] at the moment [τ]. In the fourth row I give the tonus of the muscle, i.e., the directly measured increase in length of the muscle per gramme increase of the load at the moment [τ]. Hence *the tonus is the directly accessible quantity, the value of which is not influenced by any aprioristic considerations.* The fifth row contains the quantity $C_2\ \left(a - \frac{\Delta L}{\Delta P}\right)$, and the sixth row the quantity lgn. $C_1\ (P - \triangle)$. According to equation IC, these two quantities ought to be equal. The seventh row contains the difference between the quantities of rows 5 and 6.

$\triangle$ is the threshold value of the stimulus, which is always determined by means of a simple graphic extrapolation. The constants C_1 and C_2 are determined in such a way that equal worth is attributed to each observation upon the value of these constants.

Experiment 2 [*Table III, fig.* 1], *December* 6, 1899.—The conditions of this experiment are the same as of Experiment I. Table XXXII contains the measurements of the ninth extension curve.

Experiment 3 [*Table VIII, fig.* 2], *November* 11, 1899.—In this case the cord was cut through between the first and second vertebræ. Table XXXIII contains the measurements of the fourteenth extension curve about one hour after the transection of the spinal cord. We conclude from these experiments *that within the limits of experimental error formula IC represents the relation between the increase of the weight and the decrease of the tonus.*

This relation being fixed, I calculated "*a*" with the aid of the integrated formula II, using the constants Δ, C_1 and C_2 of Tables XXXI, XXXII and XXXIII.

TABLE A		TABLE B		TABLE C	
τ	a	τ	a	τ	a
Sec.		Sec.		Sec.	
3·0	20×10^{-3}	3·0	$17·3 \times 10^{-3}$	3·0	$20·4 \times 10^{-3}$
6·2	21·2	6·2	20·7	6·2	20·4
12·6	19·7	12·6	20·4	12·6	19·1
25·4	17·7	25·4	18·6	25·4	16·7
33·5	16·8	35·0	17·6	30·9	16·0

We conclude, therefore, *that "a" is not constant.* The deviation of a from constancy induced me to investigate the beginning of the extension curve, especially the interval 0 sec. to 3 sec.

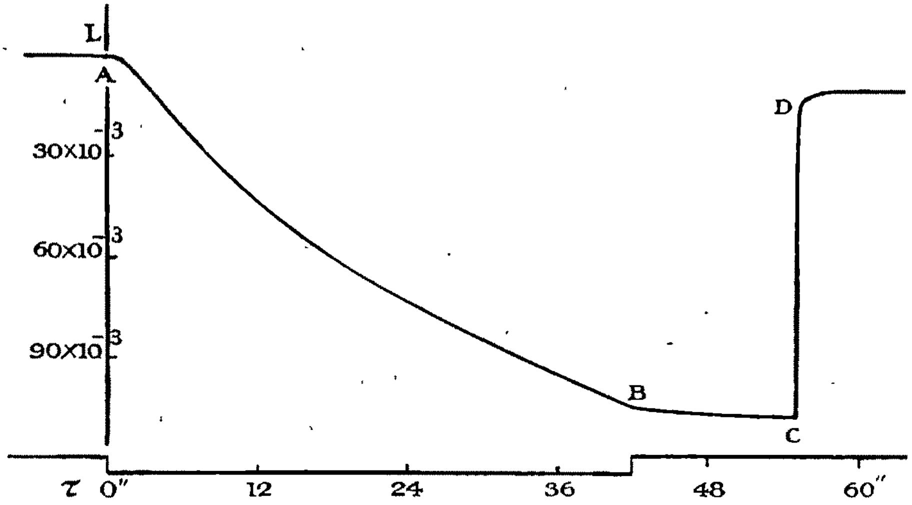

FIG. 31.—Length-load diagram of the tonic muscle.

Experiment 4 [*Table I*], *November* 10, 1899.—The frog used for this experiment was intact and wrapped up in cotton-wool. Table XXXIV contains the measurements of the thirteenth extension curve, and fig. 31 is the graph of the length-load diagram. Along the abscissa the time is noted in seconds, and along the ordinate the increment in length of the muscle in centimetres. Because the weight increases uniformly with the time, the curve ABCD is a length-load diagram. At A the inflow of the mercury in the recipient begins and the muscle lengthens under the

influence of the increasing weight. At B the inflow is stoppe
muscle from that moment onwards under the influence of a con
slowly continues to lengthen. At C the muscle is abruptly
and shortens, at first quickly till D, and then slowly cre
After a short interval of rest the cycle is repeated.

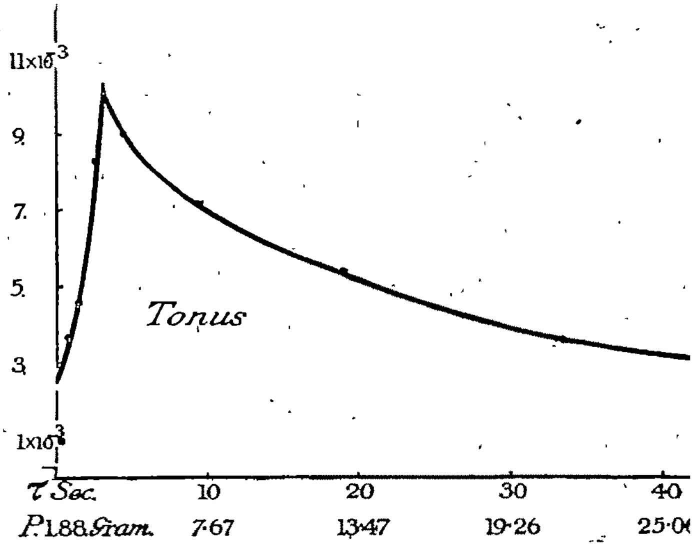

Fig. 32.—Tonus diagram of Experiment 4.

Fig. 32 is the tonus diagram. It appears from this g
the tonus at first quickly increases and then decreases. The
increasing tonus lasts in this case about 3 sec., and the
decreasing tonus 40 sec. The initial and rapid increase of
under the influence of the increasing weight is a fact fou
the following experiments. We conclude, therefore, *that*
influence of a uniformly increasing weight the tonus at fir
increases and then slowly decreases. For the interval of
tonus the relation IC holds good, as may be seen from Tabl
If we calculate *a* by means of formula II we find that
increases and then decreases. According to our definition
quantity is the sum of an elasticity and of a plasticity, and th
now arises which of these two quantities varies. There
moments at which the elasticity of the muscle may be d
directly from the length-load diagram. Firstly, the elasticit
determined in the neighbourhood of A (fig. 31), and I choos

purpose the interval 0·5 sec. to 1 sec. The interval 0 sec. to 0·5 sec. is in most cases not suitable for the determination of the elasticity, because passive resistances, residing in the apparatus and probably also in the muscle, oppose themselves to the extension of the muscle. The elasticity, as determined from that interval, is therefore evidently too small. At the interval 0·5 sec. to 1 sec. the passive resistances no more make their influence felt, and the charge is still small, so that the after-phenomenon vanishes against the elastic elongation. In the second place we may determine the elasticity from the part CD (fig. 31), because CD is the shortening caused by a known and abrupt variation of the charge. The position of D, however, where the direct elastic shortening goes over into the after-phenomenon, is not quite fixed, but this uncertainty is largely counterbalanced by the advantage that the simultaneous variation of the length and the load are fairly considerable. At the moment the experiments were made, I could not foresee that this part of the tracing would be of interest, so that a certain number of my tracings are not suitable for the determination of the elasticity from the part CD. I found, however, in my material a sufficient number of tracings which allowed of the determination of the elasticity also from that part of the curve. The result of the determination of the elasticity from both parts of the tracing is that the elasticity, as determined from the beginning and from the end of the tracing is the same in both instances. We conclude, therefore, *that the elasticity is constant and that the variation of a is due to a variation of the plasticity of the muscle.* This fact agrees with the results obtained in the first section of this research. There I found that the elasticity of the atonic muscle plays only a subordinate rôle, inasmuch as the elasticity varies but little and the problem is principally one of plasticity. It seems also in harmony with the results of modern physiology, which considers muscle-contraction chiefly as a process of deformation.

The plasticity of the tonic muscle is now determined in the following manner. First the constants Δ, C_1 and C_2 are fixed by means of formula IC. Secondly, $[a\ P]$ is calculated by introducing the values of these constants in formula II. Next, the elasticity is determined from the beginning and the end of the length-load diagram. If we keep in view that a is the sum of an elasticity and of a plasticity, we may determine the plasticity by introducing the value of the elasticity in the expression $[a\ P]$.

Fig. 33 is the plasticity diagram. I noted in this diagram the time along the abscissa and the plasticity along the ordinate. The diagram shows that the plasticity of the muscle as well as the tonus at first quickly increase and then slowly decrease. When the inflow of the mercury is stopped the stimulation of the proprioceptors ceases, and the plasticity falls suddenly.

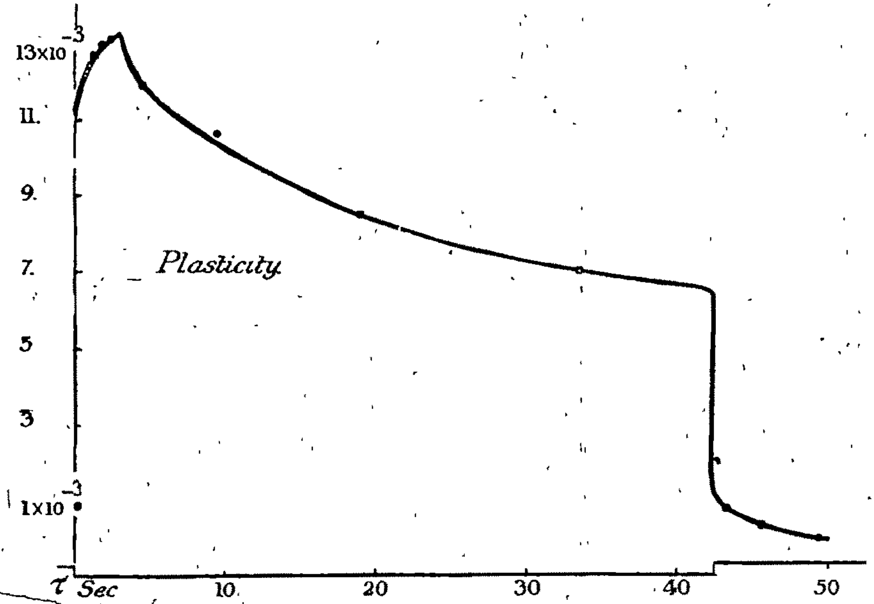

FIG. 33.—Plasticity diagram of Experiment 4.

Experiment 5 [*Tables IX, X*], *December* 2, 1899.—The frog used for this experiment was intact and wrapped up in cotton-wool. I recorded first a number of extension curves of the tonic muscle, and then I cut through the nervus tibialis. By the section of the nerve the proprioceptive reflex arc is broken, and the muscle becomes accordingly atonic. About half a minute after the section of the N. tibialis I began to record a set of extension curves of the atonic muscle. Table XXXVI contains the measurements of the last extension curve of the tonic muscle before the section of the nerve. The constants Δ, C_1 and C_2 were determined from the interval 3 sec. to 25 sec., and Table D shows how far the measurements agree with formula IC.

Fig. 34 represents the tonus diagram and figure 35 the plasticity diagram.

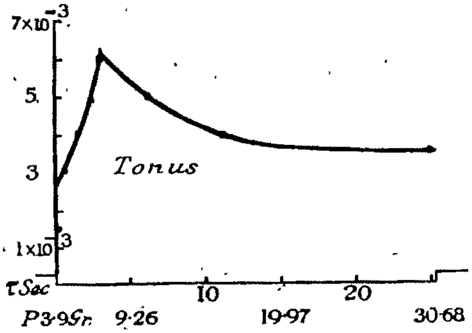

FIG. 34.—Tonus diagram of Experiment 5.

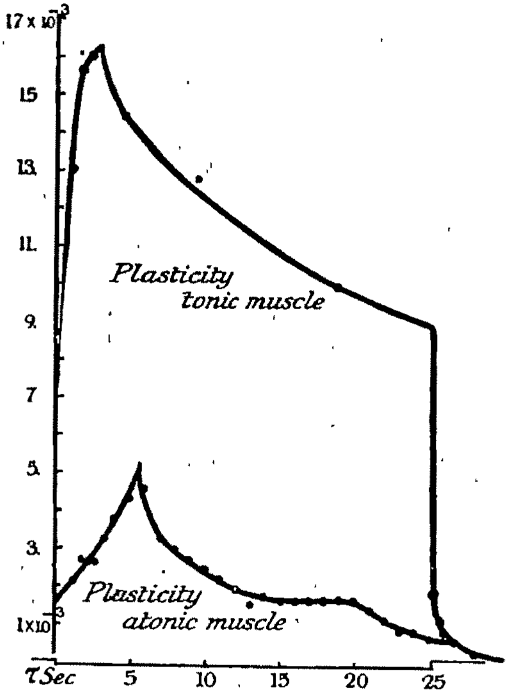

FIG. 35.—Plasticity diagram of Experiment 5.

TABLE D.

τ	$C_2\left(\alpha - \frac{\Delta l}{\Delta P}\right)$	lgn. $C_1(P - \Delta)$	Diff.
Sec. 3·0	$-$ 0·720	$-$ 0·717	+ 0·003
6·2	$-$ 0·600	$-$ 0·570	+ 0·030
12·6	$-$ 0·480	$-$ 0·481	$-$ 0·001
25·0	$-$ 0·420	$-$ 0·429	$-$ 0·009

$\Delta = 0{\cdot}3$ $C_1 = 7{\cdot}74 \times 10^{-3}$ $C_2 = -120$

The N. tibialis was cut through 45·2 sec. after the beginning of the last extension curve of the tonic muscle, the muscle being loaded at that moment with 30·9 grm. The interval immediately following the section of the nerve, from 45·2 sec. to 50 sec., is irregular, the course of the experiment being broken by contractions, but from 50 sec. to 65 sec. these contractions have ceased, and it is now possible to determine the course of the after-phenomenon. It appears from the comparison of the after-phenomenon before and after the section of the nerve that by the section of the nervus tibialis the after-lengthening is activated for a short while,[1] or in other words, that the interruption of the proprioceptive reflex arc exalts for a moment the plasticity of the muscle. The muscle is unloaded about 20 sec. after the section of the nerve. The experiment was so conducted that it was possible, without any doubt, to determine the shortening of the muscle caused by the abrupt diminution of the charge by 27 grm. The elasticity amounted in this case to $3{\cdot}09 \times 10^{-8}$, being the same as at the beginning, where it was $3{\cdot}04 \times 10^{-8}$. We see, therefore, that the section of the motor nerve affects immediately the plasticity of the muscle, but not the elasticity. The first extension curve of the atonic muscle is recorded about 25 sec. after the section of the nervus tibialis. In fig. 35 I reproduce the plasticity diagram derived from this extension curve, drawn on the same scale as the plasticity curve deduced from the immediately foregoing extension curve of the tonic muscle. It is evident from fig. 35 that by the interruption of the proprioceptive reflex arc, the plasticity of the muscle is considerably lowered.

Experiment 6 [*Table XI, fig. 3*], *December* 3, 1899.—This experiment is exactly of the same kind as No. 5.

Table XXXVII contains the measurements of the last extension curve of the tonic muscle and the effect evoked by the section of the nervus tibialis. Table XXXVIII reproduces the measurements of the first and second extension curves after the section of the nerve. The constants Δ, C_1 and C_2 are determined from the interval 3 sec. to 25·4 sec., and Table E shows the agreement with formula IC.

Figs. 36 and 37 are the reproductions respectively of the tonus and the plasticity diagrams.

The N. tibialis was divided 40·6 sec. after the beginning of the last extension curve of the tonic muscle, the muscle being loaded at that moment with 28·09 grm. It was possible in this case to determine the course of the after-phenomenon during the interval following the section of the nerve. The measurements carried out upon that part of the curve falling within the interval 45 sec. to 56 sec. show that the section of the nerve strongly accentuates the process of after-lengthening. The quantity $\left[\frac{\Delta l}{\Delta \tau} \times \tau\right]$, if the moment at which the nerve is divided is taken as the zero of time, is, however, not constant, but at first increases and then decreases, probably striving towards constancy.

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[1] A. Benedicenti (*Arch. ital. d. Biol.*, 1897, vol. xxviii) also arrived at the result that the section of the nerve is followed by a permanent lengthening of the muscle.

TABLE E.

τ	$C_2 \left(a - \frac{\Delta l}{\Delta P}\right)$	lgn. $C_1(P - \Delta)$	Diff.
sec.			
3·0	− 0·851	− 0·857	− 0·006
6·2	− 0·709	− 0·705	+ 0·004
12·6	− 0·568	− 0·565	+ 0·003
25·4	− 0·426	− 0·429	− 0·003

$\Delta = 0{\cdot}4$ $C_1 = 4{\cdot}24 \times 10^{-3}$ $C_2 = -129$

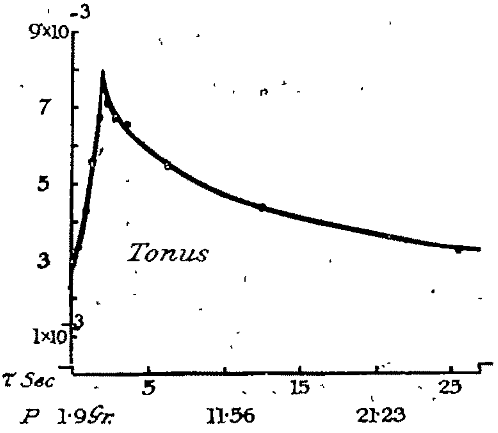


FIG. 36.—Tonus diagram of Experiment 6.

The muscle was unloaded about 27 sec. after the section of the nerve and the elasticity determined at that moment amounted to $3{\cdot}57 \times 10^{-3}$. The elasticity at the beginning of the last extension curve of the tonic muscle, i.e., before the section of the nerve, amounted to $3{\cdot}36 \times 10^{-3}$. We see, therefore, that the section of the motor nerve immediately alters the value of the plasticity of the muscle, but that the value of the elasticity remains at first constant.

The first extension curve of the atonic muscle was recorded about 72 sec. after the section of the nerve. The elasticity of the muscle determined from the beginning of this curve amounted to $2{\cdot}16 \times 10^{-3}$, and 84 sec. later, at the beginning of the second extension curve of the atonic muscle, the value of the elasticity is $2{\cdot}09 \times 10^{-3}$. The elasticity, therefore, shortly after the section of the motor nerve also diminishes, but, as seen from Table XXXVIII, the diminution of the plasticity following the initial period of exaltation is the most striking effect of the interruption of the proprioceptive reflex arc.

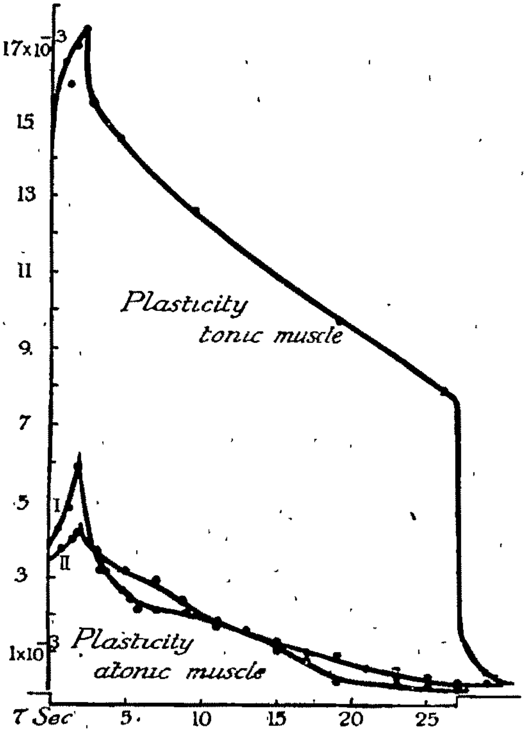


FIG. 37.—Plasticity diagrams of Experiment 6.

Experiment 7, *December* 1, 1899.—This experiment is of the same kind as Nos. 5 and 6, but the section of the motor nerve took place after the muscle was unloaded.

Tables XXXIX and XL contain the measurements respectively of the last extension curve of the tonic muscle and of the first and second extension curves after the section of the N. tibialis. Figs. 38 and 39

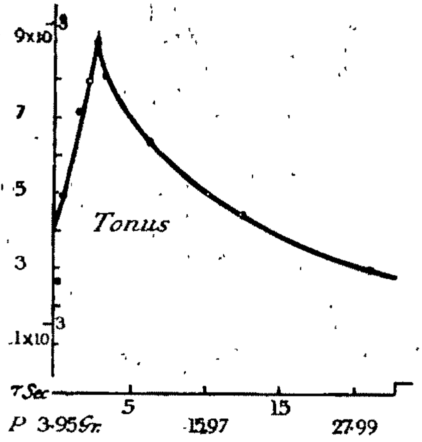


FIG. 88.—Tonus diagram of Experiment 7.

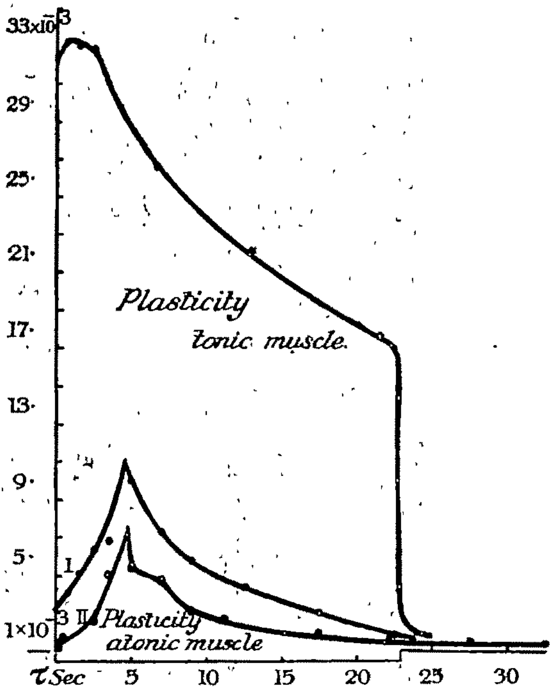


FIG. 89.—Plasticity diagrams of Experiment 7.

are the pertaining graphs. The constants Δ, C_1 and C_2 are determined from the interval 3 sec. to 22 sec., and Table F shows in how far this part of the curve agrees with the formula IC.

TABLE F.

τ	$C_2\left(\alpha - \frac{\Delta l}{\Delta P}\right)$	lgn. $C_1(P - \Delta)$	Diff.
Sec.			
3·0	− 0·600	− 0·597	+ 0·003
6·2	− 0·458	− 0·452	+ 0·006
12·6	− 0·316	− 0·314	+ 0·002
22·0	− 0·213	− 0·217	− 0·004

$\Delta = 0·3$ $C_1 = 1·27 \times 10^{-8}$ $C_2 = -70·5$

The elasticity, as determined from the beginning of the last extension curve of the tonic muscle, amounted to $2·39 \times 10^{-8}$. The value found by the unloading of the muscle is $2·66 \times 10^{-8}$, but this last determination was not quite certain.

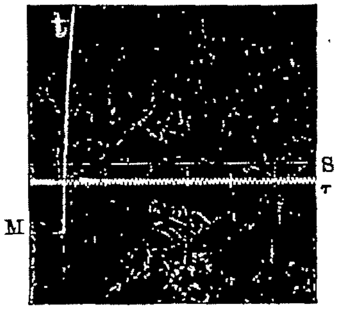


FIG. 40.—Myograph showing the after-lengthening of the M. gastrocnemius immediately after the section of the N. tibialis. M, the myograph; S, horizontal line drawn by the signal; t, the twitch evoked by the section of the nerve; τ, the time marked in tenths of seconds. After the contraction the line of the myograph runs through the time marking. By the reproduction this part of the myograph has become very indistinct, but in the original tracing the line was distinctly to be seen.

A short while after the unloading the muscle arrived at a state of rest, the length being constant; then the motor nerve was cut through, the muscle being loaded at that moment with only 3·95 grm. The section of the nerve is accompanied by a maximal contraction, after which the muscle does not return to its original length (fig. 40).

Immediately after the contraction the muscle begins to lengthen, and this lengthening exhibits all the characteristics of an after-phenomenon. It appears from the form of the after-lengthening curve that the muscle does not again reach its original length, but tends to a state of rest, at which its length is less than it was before the section of the nerve.

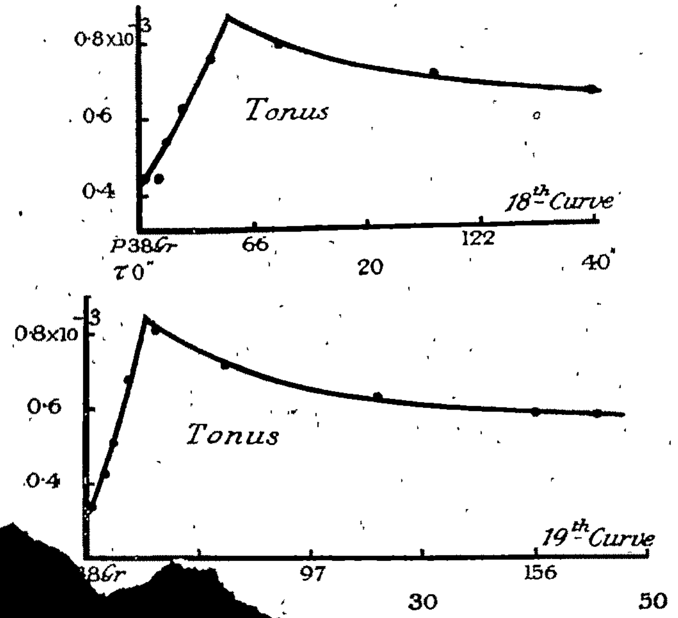
This experiment elucidates the much-discussed question, whether a tonic muscle after the section of its motor nerve becomes longer or shorter. It is evident that what happens after the section of the nerve depends chiefly upon the external conditions. For the section of the nerve acts as a supramaximal stimulus, which provokes a contraction, and meanwhile a permanent deformation (*vide infra*, Chapter III). When the load is heavy enough to eliminate this deformation and to reduce the muscle to its initial length, the muscle will become longer than
before by the activation of the after-lengthening. When th
sufficient to neutralize the permanent deformation, th es in the
the after-lengthening will in most cases not suffice first extension
shortening caused by the permanent deformatio
muscle will be shorter after the section of its mot from the end

I determined in this case also the value of the qu $^{-8}$ deter-
and found that this quantity at first increases and then decrea the
same manner as in the preceding experiment.

The elasticity determined from the beginn the
curve of the atonic muscle amounts to
of the curve at the moment of the
mined from the beginning of the sec
value of the elasticity 2·40 × 1
curve, 2·43 × 10^{-3}. There
or nerve a sl

of this research, the elasticity may again slightly increase *pari passu* with the disappearance of the plasticity.

The next group of experiments was made with the M. triceps suræ of a cat, whose spinal cord was previously transected. Full-grown,



diagrams of Experiment 8.

nd extension curve I found fo

^{3}, and from the end of the same

also in this case shortly after the section of the moto ight diminution of the elasticity, but the fall of the plasticity is the prevailing result of the interruption of the proprioceptive reflex arc. We conclude, therefore, from these experiments *that the section of the motor nerve, by which the proprioceptive reflex arc is broken, exerts a twofold effect upon the striped muscle, viz., it immediately activates, though only for a few seconds, the after-lengthening; this rise of plasticity is followed by a considerable fall, which is permanent*, and may, under conditions described in the first part of this research, lead to the complete loss of the plasticity; *it leaves the elasticity at first unaltered, but within half a minute the elasticity also diminishes. The diminution of the elasticity, however, does not seem to be very considerable*, and, as, described in the first part

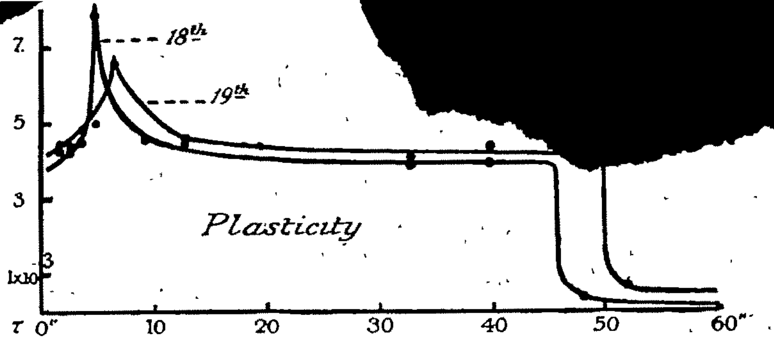


FIG. 42.—Plasticity diagrams of Experiment 8.

strong cats proved to be the most suitable for the experiments. The operation was performed under ether narcosis, but after the transection of the spinal cord no more ether was given. The transection of the cord was always followed by spinal shock, but after about one hour the muscles partially regained their tonicity. The technique of these experiments is more difficult and more complicated than in the case of the frog, so that the experimental results are less exact. The methods however, were the same.[1]

The extension curve of the tonic M. triceps of the hind limb of the spinal cat exhibits exactly the same character as those of the frog, and the analysis of the tracings is based upon the same principle.

Experiment 8 [*Table XVIII*], *December* 4, 1900.—The spinal cord of the cat was transected at 11.30 a.m. between the last cervical and the first thoracic vertebræ. In the beginning of the experiment the extension curves are strictly linear, but as soon as the shock passes away the curves assume gradually the form characteristic for the tonic muscle. Tables XLI and XLII contain the measurements respectively of the eighteenth and nineteenth extension curves. The eighteenth is recorded at 1.23 p.m., and the nineteenth curve about three minutes later. Fig. 41 represents the tonus diagrams, and fig. 42 the plasticity diagrams.

Experiment 9 [*Table VII*], *December* 6, 1900, is of exactly the same kind. The transection of the spinal cord was performed between the sixth and seventh thoracic vertebræ at 10.30 a.m. The first extension curves are linear, but already the seventh curve shows a slight deviation from the straight line. Tables XLIII and XLIV contain the measurements of the fifteenth and sixteenth extension curves. The fifteenth curve was recorded at 1.48 p.m., and the sixteenth curve was registered about three minutes later. Figs. 42A and 43 are the tonus and the plasticity diagrams respectively.

We conclude from these experiments *that the statements made for the tonic muscle of the frog hold good also for the tonic muscle of the spinal cat*. There are, however, differences between the tonic muscle of the frog and that of the cat. The most striking difference resides in the form of the plasticity diagram. In the cat the increase of the plasticity takes place more abruptly, and also the subsequent fall is more sudden, until a level is reached, which remains nearly constant during the increment of the load. Moreover, in the cat the plasticity still more prevails over the elasticity than is the case in the frog.

The following group of experiments consists in making extension curves of the normal human triceps suræ with the myotonometer of

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[1] A detailed description of the technique of these experiments is given in *Archiv. f. Physiol.* Jahrgang 1902, S. 243.

Mosso [33]. Experiments with the triceps suræ of the normal cat, i.e., without previous transection of the spinal cord, could only be done when the cat was under ether narcosis. The results obtained under

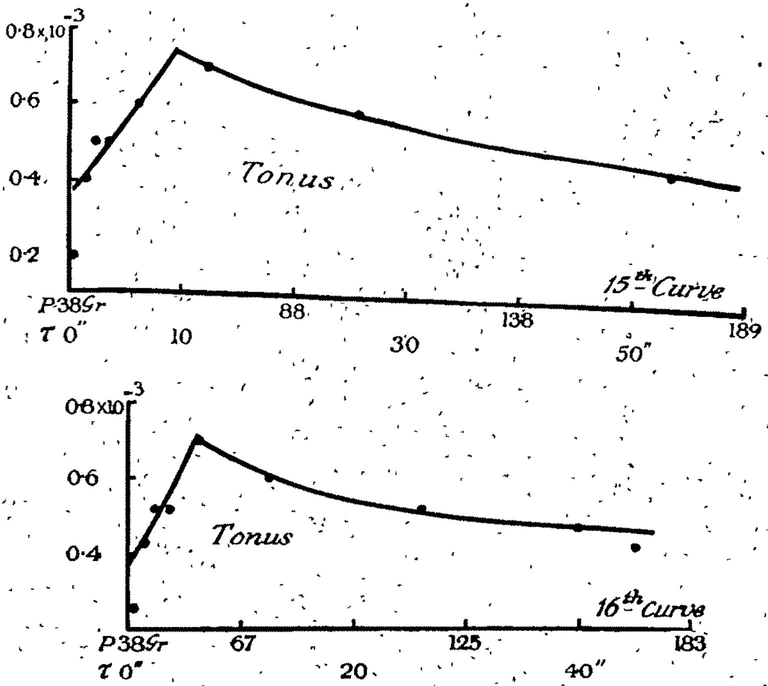

FIG. 42A.—Tonus diagrams of Experiment 9.

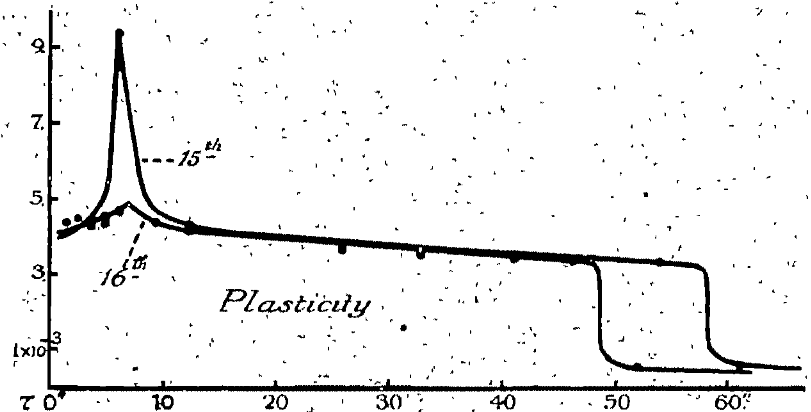

FIG. 43.—Plasticity diagrams of Experiment 9.

these experimental conditions are fully discussed in *Engelmann's Archiv*, Jahrgang 1902, and I will only quote here the conclusion. I concluded from these experiments *that probably the higher centres,* Mosso [33]. Experiments with the triceps suræ of the normal cat, i.e., without previous transection of the spinal cord, could only be done when the cat was under ether narcosis. The results obtained under these experimental conditions are fully discussed in *Engelmann's Archiv*, Jahrgang 1902, and I will only quote here the conclusion. I concluded from these experiments *that probably the higher centres,*

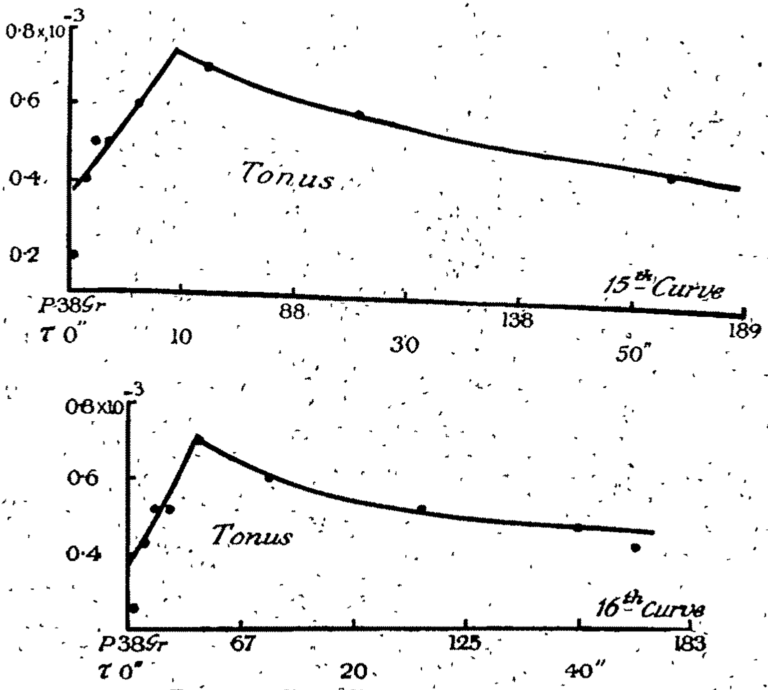

FIG. 42A.—Tonus diagrams of Experiment 9.

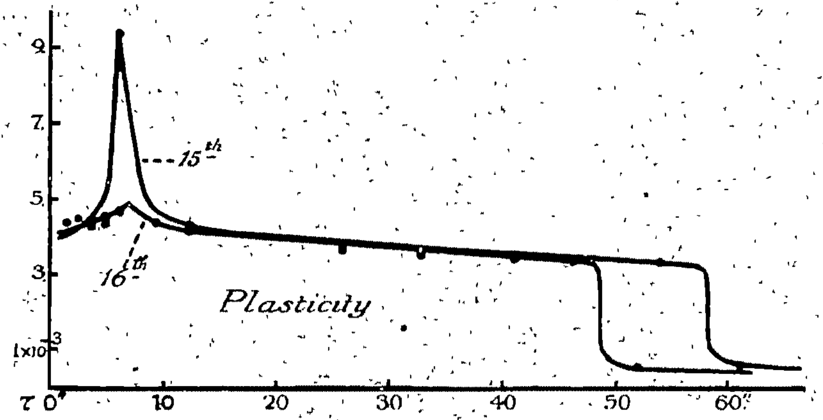

FIG. 43.—Plasticity diagrams of Experiment 9.

The values given in the tables represent by approximation the real increment of the load and the corresponding real increment of the length of the muscle. These values are obtained by multiplying the increment of the load on the scale-pan and the increment of the length

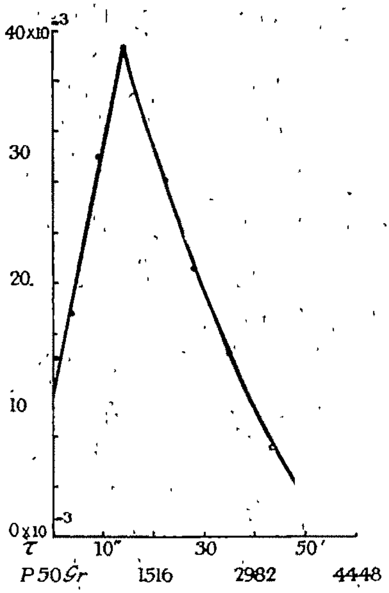

FIG. 46.—Tonus diagram of Experiment 11.

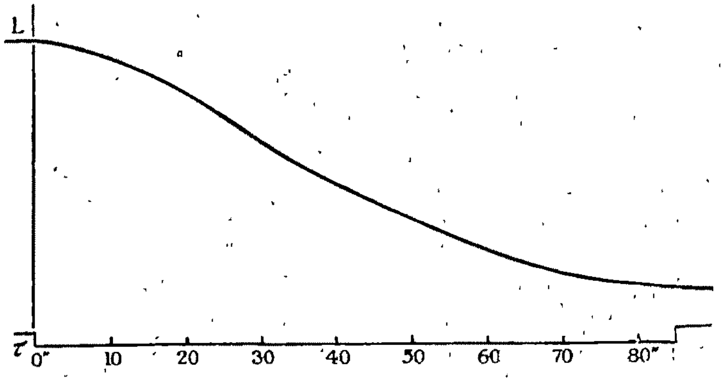

FIG. 47.—Length-load diagram of the tonic M. triceps suræ of B.

as recorded on the drum each by a coefficient deduced from the constants of the instrument and from the dimensions of the foot.

Experiment 10, *April* 6, 1902.—Triceps suræ of L. L. belongs, from the bodily aspect, to a hereditary hypotonic type (asthenicus, Stiller). Fig. 44 is the copy of the original extension curve after elimination of the respiratory movements; fig. 45 is the derived tonus diagram. Table XLV contains the measurements.

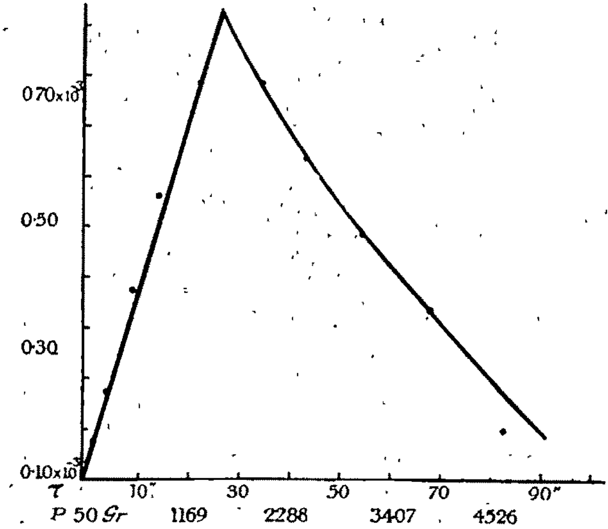

FIG. 48.—Tonus diagram derived from the extension curve of fig. 47.

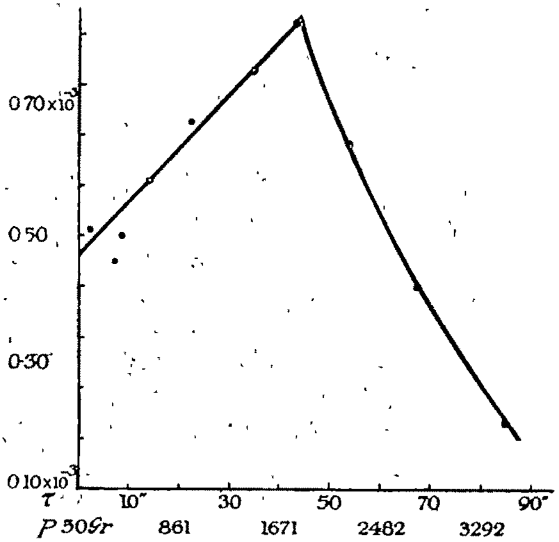

FIG. 49.—Tonus diagram of Experiment 13.

Experiment 11, *March* 16, 1902.—Triceps suræ of L. Fig. 46 is the tonus diagram, and Table XLVI contains the measurements.

Experiment 12, *April* 6, 1902.—Triceps suræ of B. B. belongs, from the bodily aspect, to a hypertonic type, with several symptoms of vagotonia (Eppinger and Hess). Fig. 47 is the rectified extension curve, fig. 48 the derived tonus diagram. Table XLVII contains the measurements.

Experiment 13.—Triceps suræ of B. Fig. 49 is the tonus diagram, and Table XLVIII contains the measurements.

The characteristic feature of these tonus diagrams lies in the lengthening of the stage of increasing tonus and the shortening of the stage of decreasing tonus. The line representing the stage of increasing tonus is probably slightly curved in the beginning but tends very soon to a straight line. For the stage of decreasing tonus we find also in this case a logarithmic relation between the increment of the load and the decrement of the tonus. I found, when determining for this part of the tonus diagram the constants C_1 and C_2—

		C_1	C_2
L.	Experiment 10	$0{\cdot}15 \times 10^{-3}$	-581
L.	,, 11	$0{\cdot}25$	-594
B.	,, 12	$0{\cdot}14$	-341
B.	,, 13	$0{\cdot}23$	-200

when approximately $\Delta = 0$. These constants are of the same order of magnitude as those in the spinal cat.

I must abstain from a further analysis of these extension curves, because my experimental material is not sufficient to trace further the rôle the elasticity and the plasticity play in the phenomenon of tonus, when the lower spinal centres are still under the control of the higher ones. We conclude, therefore, only *that the tonus diagram of the human triceps suræ resembles closely the tonus diagram of the same muscle in the normal cat when under superficial ether narcosis; that in comparison with the spinal cat the stage of increasing tonus is lengthened and the stage of decreasing tonus is shortened; that for the stage of decreasing tonus the logarithmic relation persists between the increment of the load and the decrement of the tonus.*

The experiments as yet described were all based upon the principle that the proprioceptors of a muscle are stimulated by stretching the muscle by means of an increasing weight. We may, however, also stimulate a proprioceptive reflex arc, at least its efferent part, by exciting the proprioceptors of the muscles antagonistic to those we wish to stimulate. The experiments based on this principle were exclusively made with the frog. The skin over the pretibial muscles was opened and two electrodes, connected with an induction coil, were placed very carefully and without pressure upon the muscle group formed by the M. tibialis anticus longus and M. peroneus. The amount of the stimulus caused by the induced current was chosen so as to produce a strong continued contraction of the pretibial muscles. The experiment was performed in the following way. When making an extension curve of the M. gastrocnemius, I stimulated the pretibial muscles during a certain interval of time. I chose this form of the experiment because the stretching and extending of the M. gastrocnemius puts into action its own proprioceptive reflex arc, and under these circumstances the effect of the second stimulus caused by the faradization and the contraction of the pretibial muscles is more obvious.

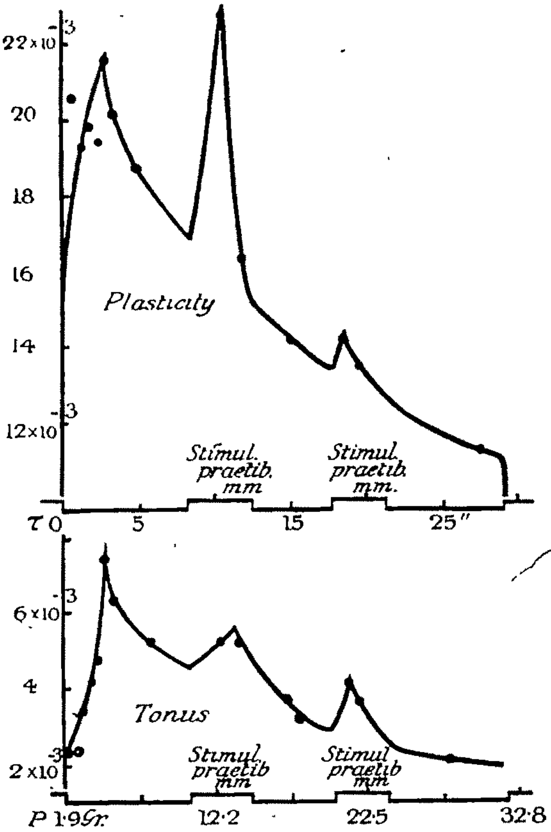

FIG. 50.—Tonus and plasticity diagrams of Experiment 14—fifteenth extension curve.

Experiment 14 [*Table XIX, X, fig.* 7], *November* 29, 1899.—The frog used for the experiment was intact and wrapped up in cotton-wool. The pretibial muscles were stimulated by an induced current, the distance of the coils being 4·5 cm. The current induced by a distance of the coils of 8 cm. was hardly felt on the tongue, and at a distance of 3 cm. the current just caused pain on the tip of the tongue.

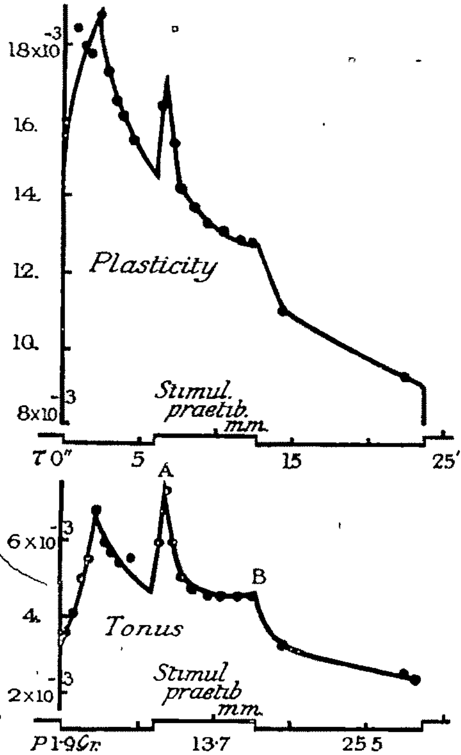

FIG. 51.—Tonus and plasticity diagrams of Experiment 14—sixteenth extension curve.

The pretibial muscles answered to this stimulation by a vigorous continued contraction. The stimulation lasted only a short while, viz., from 8·4 to 12·4 sec., and from 17·9 to 21·4 sec., reckoned from the beginning of the extension curve. Table XLIX contains the measurements of the fifteenth extension curve of the M. gastrocnemius, and fig. 50 represents the tonus and the plasticity diagrams. We see from this experiment that the stimulation of the proprioceptors of the pretibial muscles is followed by a strong increase of the tonus of the M. gastrocnemius. According to our definition of tonus, the M. gastrocnemius becomes more extensible, and this increment of the extensibility is chiefly caused by an increase of the plasticity of the muscle. The effect of the stimulation may still continue after the stimulus has already ceased. We conclude from this experiment *that the stimulation of the pretibial muscles by a faradic current, and the subsequent contraction of these muscles, cause a considerable increase of the tonicity of the M. gastrocnemius; this effect may even persist some seconds after the cessation of the stimulus. The increment of the tonus is chiefly due to an increase of the plasticity of the muscle.*

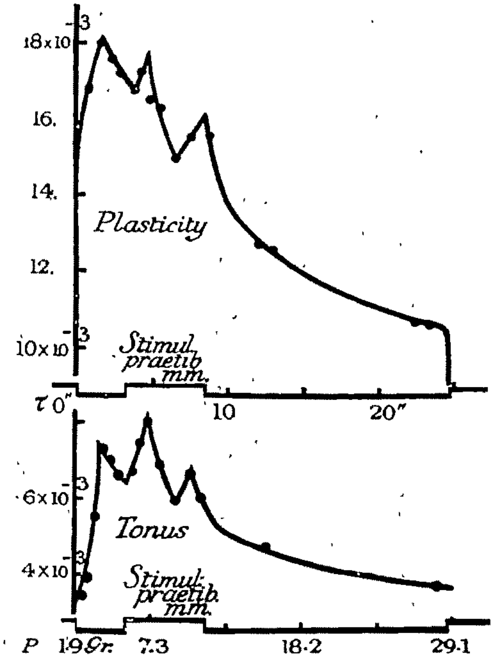

Fig. 52.—Tonus and plasticity diagrams of Experiment 15 (intact animal).

The increase in tonicity of the agonist under the influence of the contraction of the antagonist is usually called *inhibition* of the agonist by the antagonist. The rôle which these phenomena play in co-ordinate movement has been fully demonstrated by Sherrington and others.

Fig. 51 is the tonus and the plasticity diagrams of the next (sixteenth) extension curve of the M. gastrocnemius. The pretibial muscles were stimulated during the interval 5·9 to 12·8 sec., and by a distance of the coils of 4 cm. The stimulus was therefore, in this case, somewhat stronger and lasted longer than in the foregoing experiment. The effect is a little different; the increment of the tonus is followed by a fall, which is again succeeded by a slight rise of the tonus. The curve representing the effect of the stimulation of the pretibial muscles upon the tonus of the gastrocnemius shows, therefore, two tops. Because these two tops will further be of interest for the description of the experiments, I will designate the first top as the A top, and the second one as the B top. The experiments now prove that the effect of the stimulation of the pretibial muscles is always a two-topped curve, provided the stimulus be of sufficient strength and of sufficient duration.

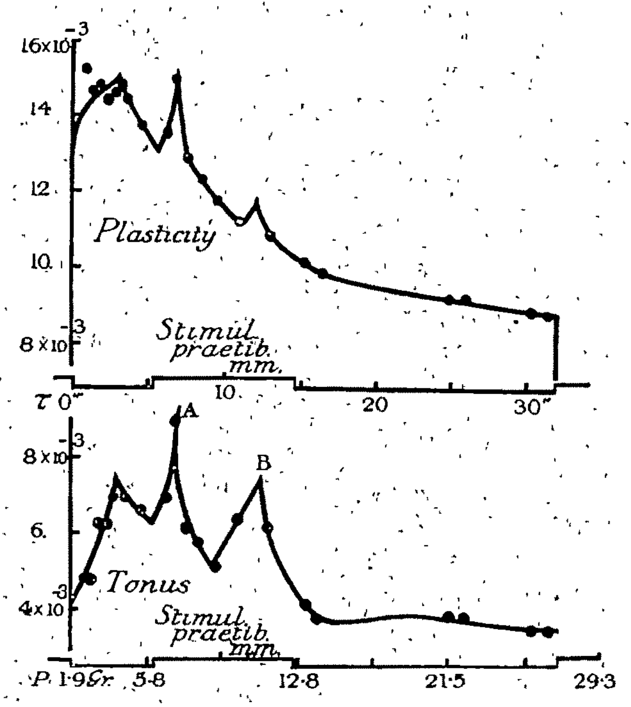

FIG. 58.—Tonus and plasticity diagrams of Experiment 15 (spinal cord transected).

Experiment 15 [*Tables XXVIII, XIX, fig.* 8], *November* 26, 1899.—This experiment was of the same kind as Experiment 14. The pretibial muscles, however, were on both sides carefully loosened from their insertions, and the distance of the coils was 5 cm. The loosening of the muscles from their insertions was done in order to avoid little movements in the joints and the concomitant stretching of the fasciæ, so that the effect of the stimulation was exclusively due to the excitation of the proprioceptors of the pretibial muscles.

Table L contains the measurements and fig. 52 the reproduction of the tonus and the plasticity diagrams. The effect of the stimulation is again a two-topped curve, and the persistence of the effect after the cessation of the stimulus is evident. In this case the B top is more marked than in the preceding experiment. The increase in the tonus always begins after a short period of latency.

Fig. 53 represents the tonus and the plasticity diagrams derived from an extension curve of the same frog, but after transection of the

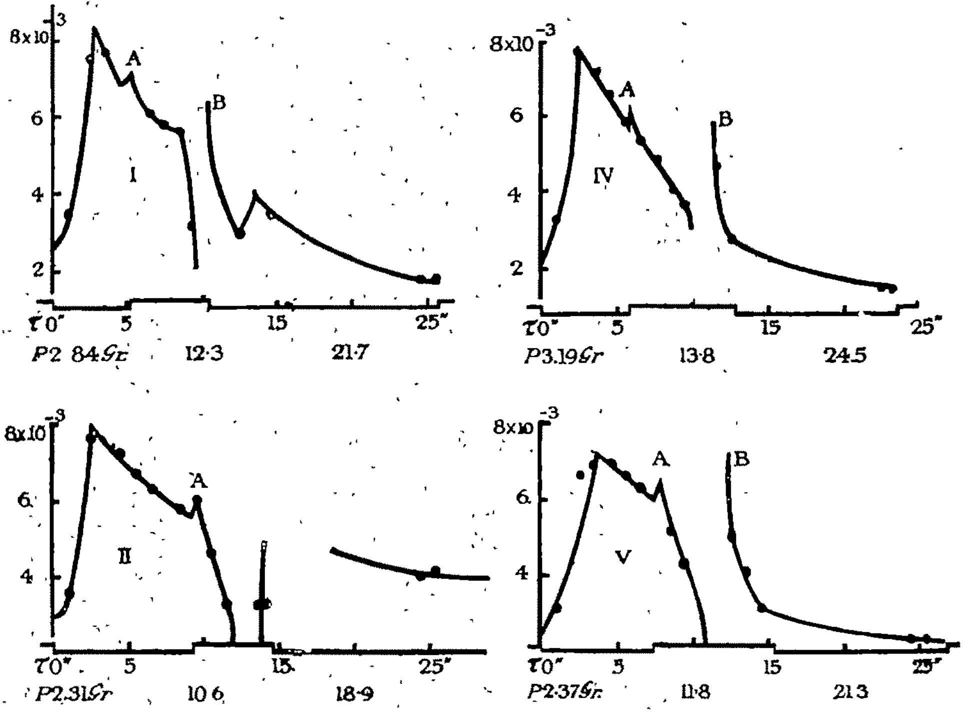

FIG. 54.—Tonus diagrams of Experiment 16.

spinal cord between the first and the second vertebræ. About one hour after the transection, the curve was recorded, being the eighth extension curve of the M. gastrocnemius after the transection of the cord. The effect is, in this case also, a two-topped curve. We conclude from these experiments that the stimulation of the pretibial muscles by a faradic current, and the subsequent contraction of these muscles, causes a considerable increase of the tonicity of the M. gastrocnemius This increase of tonicity has two maxima, one shortly after the beginning of the excitation of the pretibial muscles (A top) and another maximum in the neighbourhood of the cessation of the stimulus (B top). The second top is, however, not caused by the cessation of the stimulus, and it is only accidentally in these experiments that the B top falls in the neighbourhood of the cessation of the stimulus. The moment at which the second top appears seems only to be determined by the strength of the stimulus and the actual state of the reflex centre.

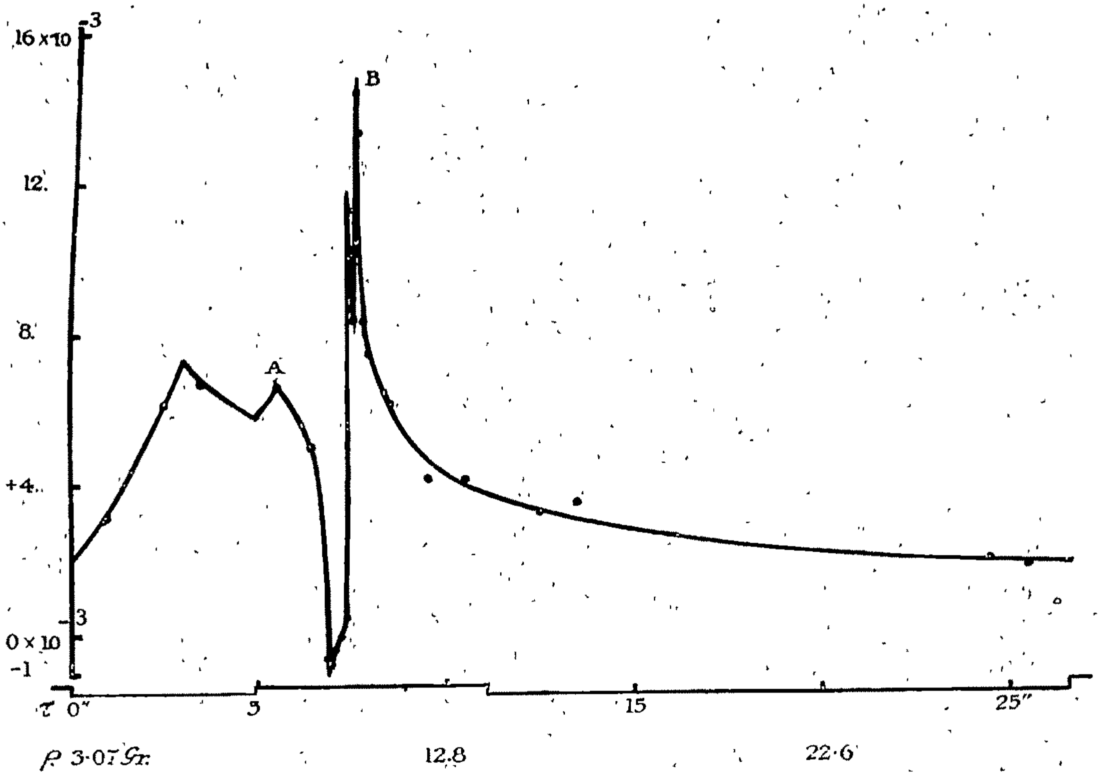

FIG. 55.—Tonus diagram of Experiment 16—third curve.

Experiment 16, *November* 25, 1899.—This experiment belongs to the same group as Experiments 14 and 15. The frog was intact, and wrapped up in cotton-wool. The pretibial muscles were loosened on either side from their insertions to avoid little movements in the joints. I reproduce in figs. 54 and 55 the tonus diagrams derived from five successive extension curves of the M. gastrocnemius. The first of these extension curves is about the fortieth curve of the whole series, recorded with intervals of approximately five minutes.

First curve (fig. 54, I).—The pretibial muscles were stimulated during the interval 5·3 to 10·4 sec., reckoned from the beginning of the extension curve, the distance of the coils being 4 cm. The initial charge amounted to 2·84 grm. and the increment of the charge to 0·94 grm. per second.

Second curve (fig. 54, II).—Interval of stimulation 8 to 14·6 sec.; distance of the coils 4 cm.; initial charge 2·31 grm.; and increment of the charge 0·83 grm. per second.

Third curve (fig. 55).—Interval of stimulation 4·9 to 11·1 sec.; distance of the coils 4 cm.; initial charge 3·07 grm.; and increment of the charge 0·98 grm. per second.

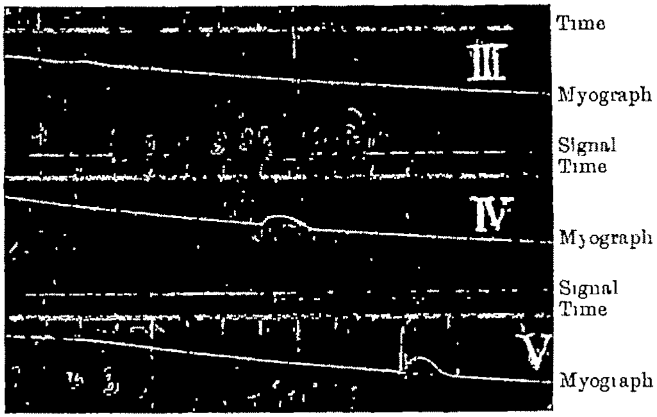

FIG. 56.—Three successive extension curves of the tonic M gastrocnemius. The curves marked III and IV show a tonic contraction evoked by the stimulation of the pretibial muscles The curve marked V exhibits, at the same time a twitch and a tonic contraction The dropping of the signal indicates the moment at which the pretibial muscles were stimulated. The enlargement is about ten times. The time in tenths of seconds

Fourth curve (fig. 54, IV).—Interval of stimulation 5·9 to 12·8 sec.; distance of the coils 4 cm.; initial charge 3·19 grm.; and increment of the charge 1·07 grm. per second.

Fifth curve (fig. 54, V).—Interval of stimulation 7·4 to 15·4 sec.; distance of the coils 4 cm.; initial charge, 2·37 grm.; increment of the charge 0·95 grm. per second.

Fig. 56 is a reproduction of the original extension curves from which the third, fourth, and fifth tonus diagrams are derived.

The five extension curves of this experiment exhibit the same general feature as those already described, but about four seconds after the beginning of the stimulation of the pretibial muscles a slight contraction appears. Jäderholm [24], who gives an ample description of this kind of contraction, designates it by the very appropriate name of "tonic contraction." The tonic contraction and the twitch are the two different forms of contraction of which the striped muscle is capable. These two forms of contraction are independent of each other, as may be seen from Curve V, fig. 56, which shows a twitch and a tonic contraction at the same time. The twitch is, without doubt, connected with the function of the striped apparatus, and therefore under the control of the motor cell of the anterior horn. The tonic contraction, however, seems to my opinion connected with the sarcoplasmatic mass, and hence controlled by the sympathetic motor cell, which innervates this part of the muscle.[1]

An interpolation shows that the twitch begins about one-tenth of a second after the commencement of the tonic contraction. The same result was found in other cases, so that we conclude *that the tonic contraction in its appearance precedes the twitch for about one-tenth of a second.* A tonic contraction of the M. gastrocnemius is more easily obtained by stimulating the proprioceptors of the pretibial muscles than a twitch, and only a repeated or continued stimulation of sufficient strength is able to elicit a twitch. We conclude from these observations *that the impulses set up in the afferent part of a proprioceptive reflex arc by stimulating the proprioceptors of a muscle not only go over upon the efferent part of its own proprioceptive reflex arc, but also upon the efferent part of those reflex arcs, which belong to muscles antagonistic to the stimulated one. The way of least resistance for the passage of the stimulus to the efferent part of an antagonistic reflex arc leads to the sympathetic motor cell.* Upon this principle is built the interaction of antagonistic muscles in the production of co-ordinated movements.

The tonic contraction may be considered as a sudden variation of the tonus, as illustrated by the tonus diagram of fig. 55. This diagram is exactly of the same form as the tonus diagrams reproduced in figs. 50, 51, 52 and 53, the only difference being that the fall of the tonus between the two tops is deeper. The difference between the diagrams, in which a tonic contraction appears, and those in which this is not the case resides, therefore, only in the depths of the tonus fall, occurring between the A and B tops. Hence we conclude that the cases in which a tonic contraction appears are continuously connected with those in which this is not the case, or, in other words, *that the tonic contraction is only a sudden and considerable variation of the tonus.*

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[1] Botazzi and Grünbaum [12] arrived at a similar result for the auricular musculature of the tortoise heart [12].

The tonus diagram of fig. 55 shows a peculiarity, viz., an accessory top between the A and B tops. In order to know if this accessory top

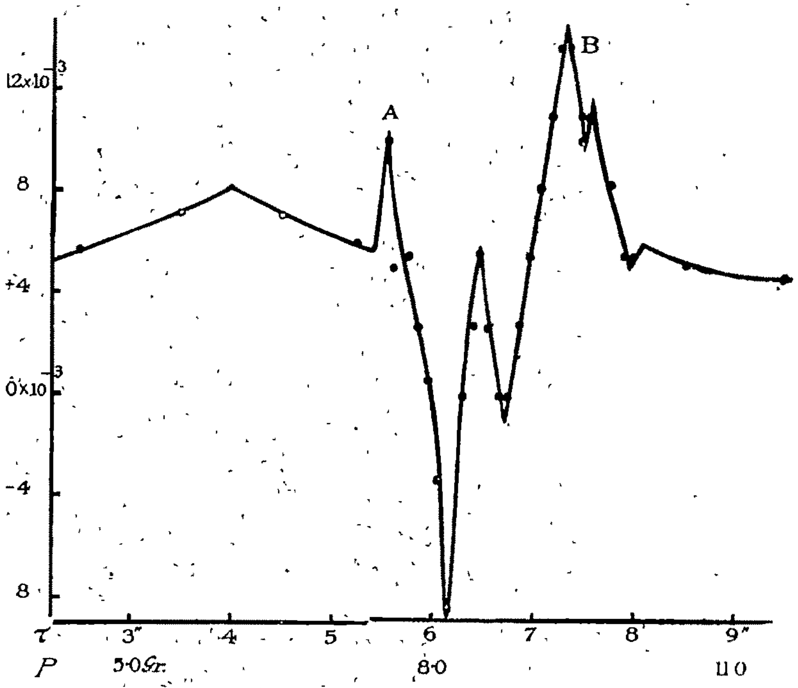

FIG. 57.—Tonus diagram belonging to Experiment 16, and showing accessory tops.

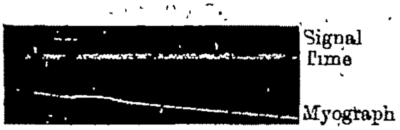

FIG. 58.—Extension curve of the tonic M. gastrocnemius, showing a tonic contraction evoked by the stimulation of the pretibial muscles. The dropping of the signal indicates the moment at which the pretibial muscles were stimulated. The enlargement is about ten times. The time in tenths of seconds.

was due to an accidental irregularity in the original tracing, I took another extension curve of the M. gastrocnemius, belonging to the same series, and also showing an apparently smooth tonic contraction. Table LI contains the measurements of this curve, and fig. 57 is the tonus diagram. The original curve is reproduced in fig. 58; there is also in this case an accessory top between the A and B tops, and above them little accessory tops upon the descending branch of the B top. The same phenomenon shows the tonus diagram of fig. 59, which is derived from the extension curve reproduced by fig. 60. This tracing belongs to another series, and the stimulation of the pretibial muscles took place at a distance of the coils of 6 cm. At first sight the tonic contraction seems smooth and continuous, but an accurate measurement of the curve reveals three accessory tops between the A and B tops. The three tops a_1, a_2, a_3, in the tonus diagram are approximately equidistant, viz., 0·75 sec., and this fact suggests the idea that the tonic contraction is a tetanus, even in those cases in which it has the appearance of a continuously smooth contraction. That this is really the case is demonstrated by Curve IV of fig. 56. The ascending branch of the tonic contraction shows clearly a staircase phenomenon. Fig. 61 shows the same phenomenon in a more marked way; this curve is the first extension curve of the series of five, of which fig. 56 reproduces the third, fourth and fifth tracings. Not only the ascending branch

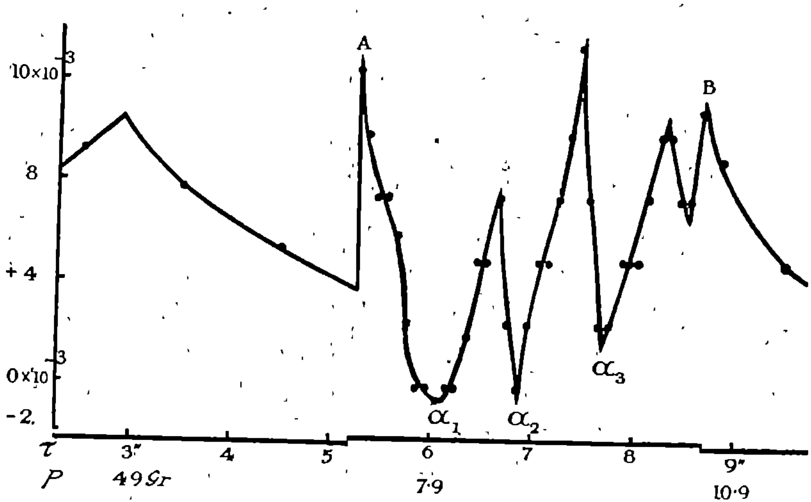

FIG. 59.—Tonus diagram showing several accessory tops between the A and the B top.

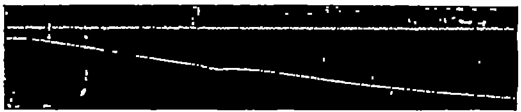

FIG. 60.—Extension curve of the tonic M. gastrocnemius showing a tonic contraction evoked by the stimulation of the pretibial muscles. The dropping of the signal indicates the moment at which the pretibial muscles were stimulated. The enlargement is about ten times. The time in tenths of seconds.

but also the descending one may exhibit the staircase phenomenon. An example of this kind shows fig. 62; this curve is the second one of the same series of five. The fact that the descending branch may also exhibit the staircase phenomenon is a further proof of the tetanic nature of the tonic contraction. We conclude, therefore, from these experiments *that the tonic contraction of the M. gastrocnemius evoked by stimulating the pretibial muscles by means of a faradic current is not a single contraction but a tetanus.*

After the tonic contraction has passed away the muscle often remains oscillating. The amplitude of the oscillation is extremely small, hardly perceptible in the tracing, notwithstanding the enlargement of the movement by the length-recorder is about tenfold. The duration of the oscillations is approximately constant, but may differ from case to case. The variation in the duration of the oscillations is about 1·5 to 2·5 sec.[1]

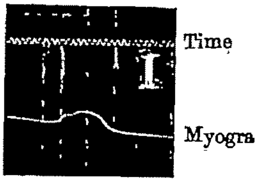

FIG. 61. — Tonic contraction of the M. gastrocnemius. The ascending branch of the contraction shows a staircase. The enlargement is about twenty times. The time in tenths of seconds.

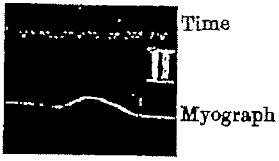

FIG. 62. — Tonic contraction of the M. gastrocnemius. The ascending as well as the descending branch shows a staircase. The enlargement is about twenty times. The time in tenths of seconds.

Curve III, fig. 56, shows four of these oscillations, beginning shortly after the tonic tetanus has finished. The duration of each oscillation is about 1·6 sec.

The curves representing the tonic contraction of the M. gastrocnemius of the frog resemble closely the contraction curves of smooth muscles published by Engelmann, Winkler, Schultz, and others. The tetanic character of the tonic contraction, the staircase phenomenon, the oscillations following the tonic tetanus, the slowness of the contraction and its little height are all the same. I conclude, therefore, *that the tonic contraction and the phenomena allied with it are due to the sarcoplasmatic part of the striped muscle. Hence we come from the physiological side also to the conception of the duality of the striped muscle, viz., a*

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[1] The same oscillatory movements are described by Ph. Botazzi and others in the case of smooth muscles of Evertebrata [11] and [12].

sarcoplasmatic mass innervated by a sympathetic motor cell, which in its function closely resembles the smooth muscles and a striped apparatus, termination of the motor cell of the anterior horn, embedded in this sarcoplasmatic stroma.

Keith Lucas [29], in his experimental work, arrived at a similar result, viz., that the muscle is composed of three different contractile substances. In my opinion his general conclusion that there are different contractible substances in the striped muscle is true, but my experiments tend to the conclusion that there are only two, in agreement with the morphological researches of Boeke.

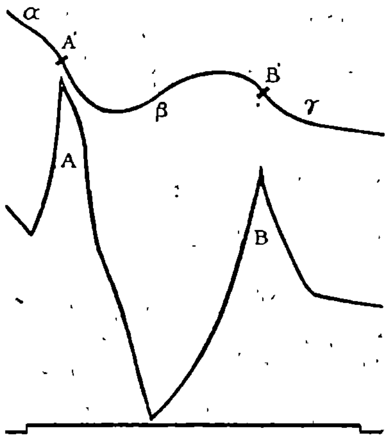

FIG. 63.—Scheme representing the effect of the stimulation of the pretibial muscles upon the form of the extension curve of the M. gastrocnemius. The curve $\alpha\beta\gamma$ is the extension curve, and the curve AB the derived tonus diagram.

The curve representing the effect of the stimulation of the pretibial muscles upon the extensibility of the M. gastrocnemius is S-shaped. Fig. 63 represents this effect diagramatically. The tonus diagram AB, derived from the extension curve $\alpha\beta\gamma$, is constructed on the supposition that the load augments uniformly as in our experiments. The temporary lengthening of the muscle represented by the part $\alpha\beta$ is usually called "inhibition," and the subsequent shortening of the muscle "tonic contraction." Inhibition and tonic contraction are two phases of the same process evoked by the stimulation of the proprioceptors of an antagonistic reflex arc. A good instance of such an S-shaped curve shows fig. 60.

The form of the $\alpha\beta\gamma$ curve is extremely variable in different experiments; it is determined by the anatomical structure of the reflex centre, by the actual state of this centre, and by the nature and the strength of the stimulus. The point *a* indicates the moment at which the inhibition begins. The determination of the exact position of this point upon the curve is only possible in the tracings of figs. 60, 64 and 65. In these cases the latent period between the beginning of the stimulation and the beginning of the inhibition varies between 0·05 and 0·1 sec. The A top of the tonus diagram coincides with the first inflection point [A′] of the extension curve, and the B top with the third inflection point [B′], situated upon the descending branch of the tonic contraction. The point *β* indicates the moment at which the tonic contraction begins. This point may easily be determined in the tracings. The five successive tracings belonging to Experiment No. 16 give the following result:—

Number	Moment at which the stimulation of the pretibial muscles begins	Beginning of tonic contraction	Difference in seconds
I	5·3	9·7	4·4
II	8·0	12·0	4·0
III	4·9	6·8	3·9
IV	5·9	9·9	4·0
V	7·3	10·5	3·2

These five experiments were made under similar conditions, and repeated with intervals of about five minutes and with the same strength of stimulus. We see from this table that the tonic contraction begins about four seconds after the commencement of the stimulation of the pretibial muscles, but that there are slight deviations from this mean in accordance with the reflex nature of the whole phenomenon. In the same way the point may be determined at which the tonic contraction has ceased. The table gives the relation between the end of the tonic contraction and the cessation of the stimulus:—

Number	End of the tonic contraction	Cessation of stimulation of the pretibial muscles	Difference in seconds	Duration of the tonic contraction in seconds
I	10·9	10·4	− 0·5	1·2
II	13·4	14·6	+ 1·2	1·4
III	8·0	11·1	+ 3·1	1·2
IV	11·4	12·8	+ 1·4	1·5
V	12·2	15·4	+ 3·2	1·7

It follows from this table that there is no fixed relation between the end of the tonic contraction and the moment at which the stimulus ceases. The duration of the tonic contraction is, however, approximately constant. Other experiments give similar results. Hence we conclude *that the effect following the stimulation of the pretibial muscles is chiefly determined by the actual state of the reflex centre and by the strength of the stimulus, but that the duration of the stimulation has but little influence*, in other words, *that the effect of the stimulation may be regarded as a reflex discharge.*

The inhibition of the tonus lasts about three times as long as the tonic contraction, and when the strength of the stimulus increases, the inhibitory effect becomes more and more predominant. Fig. 64 may prove this. The curve belongs to the series of Experiment 16 and was the thirteenth tracing. The distance of the coils amounted to but 3 cm., so that the stimulus in this case was rather strong. When the strength of the stimulus increases still further, the general feature of the curve remains the same, viz., S-shaped, but the tracing becomes irregular. Fig. 65 may prove this. The curve of fig. 65 is the fourteenth of the same series, and the distance of the coils amounted to 2·5 cm. We conclude from the preceding experiments *that inhibition of tonus and tonic contraction are two phases of the same excitatory process.* The inhibitory effect is only caused by the fact that the sarcoplasmatic part of the striped muscle shortly after the beginning and also near the end of its excitatory state shows an increment of its extensibility, which is due to a temporary increase of its plasticity.[1] The contraction curve of smooth muscles shows also two maxima of extensibility, one shortly after the beginning of the contraction and the other near the end, exactly in the same way as the tonic contraction

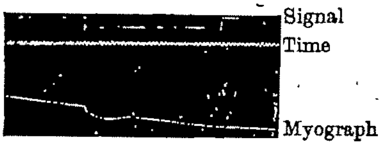

FIG. 64.—Extension curve of the tonic M. gastrocnemius showing a tonic contraction evoked by the stimulation of the pretibial muscles. The dropping of the signal indicates the moment at which the pretibial muscles were stimulated. In this case the inhibitory effect is predominant. The enlargement is about ten times. The time in tenths of seconds.

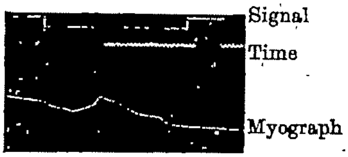

FIG. 65.—The same experiment as reproduced in fig. 64, but with a still stronger stimulus. The enlargement is about ten times. The time in tenths of seconds.

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[1] Schenck also established the fact that the extensibility of the striped muscle is increased at the beginning of the contraction (*Pflüger's Arch.*, 1900, Bd. lxxxi, S. 595).

of the striped muscles. *There exists also a close analogy between the function of the smooth muscle and the function of the sarcoplasmatic part of the striped muscle* [23].

CHAPTER III.—THE ISOTONIC CONTRACTION.

The analysis of the extension tracings of the tonic muscle in the second section of this research leads to the conclusion that the formula

$$-l = \frac{1}{C_1C_2}\left[C_1(P-\Delta)\langle \mathrm{lgn}.C_1(P-\Delta) - 1 \rangle\right]$$

represents the shortening of the muscle [$-l$] caused by the stimulation of the proprioceptors through a uniformly increasing weight [P]; this result, however, could not be tested directly. I tried, therefore, to prove that the formula quoted above represents the relation between the amount of the stimulus in physical units and the lift of the muscle when working under isotonic conditions. The conditions under which the muscle is contracting in the two cases are not strictly the same, but these differences will only affect the value of the constants of the formula, not the form of the function. Much work has been done by the earlier observers to elucidate the relation between stimulus and lift for a muscle contracting isotonically. A great deal of this work was performed by very skilful experiment, and in such an exact way that it seems for my purpose superfluous to repeat it. Hence I shall use in this section the experimental material of others.

The problem involves in the first place the question of the relation between the stimulus in physical units and the stimulus in physiological units, or whatever part of the stimulus, considered as a physical cause, is transmuted into a physiological cause. Generally it is admitted that the relation

$$\frac{\text{stimulus in physical units}}{\text{stimulus in physiological units}} = \text{constant}.$$

Now I have shown in my publication on the principle of entropy in physiology [28] that all physiological systems are endowed with passive resistances, so that the increment of the stimulus ought to surpass a certain finite limit before producing an increment of the effect. We find, therefore, that all the stimuli [P ± δ], in which [δ] means a finite increment of P, yield the same effect. The value of [δ] may be comparatively great when the passive resistances in the physiological system are considerable. It is evident from the simple recognition that all physiological systems are systems with passive resistance that the same relation

$$\frac{\text{stimulus in physical units}}{\text{stimulus in physiological units}} = \text{constant}$$

cannot exist for the whole scale of stimuli. For though $[\delta]$ is small in general in comparison with P, it will nevertheless sensibly influence the value of P when P itself is small. Hence, we conclude *that if we apply, as is usually done, the relation*

$$\frac{\text{stimulus in physical units}}{\text{stimulus in physiological units}} = \text{constant}$$

to the whole scale of stimuli, we ought to find deviations between the observed and the calculated values of $[-l]$ *in the neighbourhood of the small values of* P.

The second question involved in the problem ensues from the plasticity of the muscle. As I have proved in the first and second sections, the muscle, in consequence of its plasticity, is easily deformed by mechanical and thermical agencies. The same happens when the muscle is compelled to contract by electrical or other artificial stimuli, applied to the motor nerve. After each contraction the muscle is slightly deformed, and when this deformation surpasses a certain limit, the effect becomes manifest in the form of a slight permanent shortening of the muscle. This deformation was first systematically studied by Tiegel [43], and later more in detail by Bohr [10]. This fact is of the highest interest, because it shows that in a series of successive contractions the muscle does not remain the same object. Hence *it is impossible to represent the relation between stimulus and lift by a formula in which enters only one independent variable—viz., the stimulus.* A formula which will give a true representation of the relation between the stimulus and the lift ought to contain at least two independent variables—viz., the variable P, the stimulus, and a second variable Q, which represents the momentary state of the muscle during the experiment; or, in other words, the effect of the stimulation is not entirely determined by the value of the stimulus in physical units. Hence we conclude *that it is only possible to represent the relation between stimulus and lift by a formula containing one independent variable, if we neglect the plastic deformation of the muscle during the experiment,* or if we try to eliminate by some artifice the influence of the plastic deformation upon the effect of the stimulation.

The formula

$$-l = \frac{1}{C_1 C_2}\left[C_1 (P - \Delta) \langle \text{lgn. } C_1 (P - \Delta) - 1 \rangle\right]$$

is reduced to its simplest form when we substitute $C_1 (P - \Delta) = R$. After this substitution it assumes the form

$$- l [C_1C_2] = F(R) = R \langle M \log. R - 1 \rangle,$$

if M is the modulus. Fig. 66 is the graphic representation of the function $F(R) = R \langle M \log. R - 1 \rangle$. The first striking feature of this function is that it shows a maximum in the neighbourhood of $R = 76$, and it may be asked if this agrees with the physiological facts. The opinions about this point are divided, and a great many observers, such as Tigerstedt, believe that the lift approaches asymptotically a limiting value, when the stimulus increases. In my opinion this conclusion is erroneous, because it is left out of consideration that the muscle is so easily deformed by a strong stimulus. It seems, therefore, to me that the asymptotical increment of the lift is not caused by a further real increase of the lift, but only by a shortening of the muscle, caused by plastic deformation. I agree, therefore, with Waller and others, who believe that the lift considered as a function of the stimulus is a maximum-phenomenon. It is another question, whether the decrement of the function by a further increment of R is also realized in nature. The experiments seem to indicate that this is really the case, but that the decrement of the lift by further increase of the stimulus in most cases is hidden by the plastic deformation of the muscle.

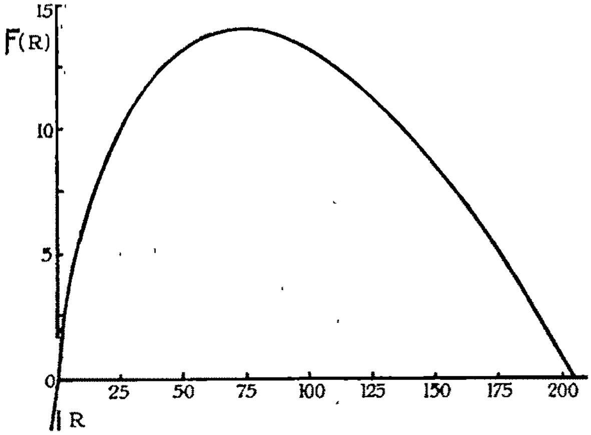

FIG. 66.—Graphic representation of the function $F(R) = R \langle M \log. R - 1 \rangle$.

Experiment 20, *of Tigerstedt and Willhard,* proves the existence of a maximum lift and shows, moreover, the plastic deformation of the muscle. For this experiment a muscle-nerve preparation of the frog is used. The nerve is stimulated by a break-induction shock. First the stimulus increases, and as soon as it reaches the value of 70, the maximum lift of 13·25 is attained; then the stimulus is gradually augmented to 75 and to 80, but the lift is only 13·15. By a further increase of the stimulus the plastic deformation becomes evident, and the lift seems to increase. When the stimulus has attained the strength of 95, the course of the experiment is reversed, and the stimulus decreases by equal steps, in the same way as it had previously increased. The curves representing the lifts as a function of the amount of the stimulus in arbitrary physical units are reproduced in fig. 67. Curve I represents the lifts by increasing stimuli, and Curve II those by decreasing stimuli. It is evident that the curves coincide at the beginning and

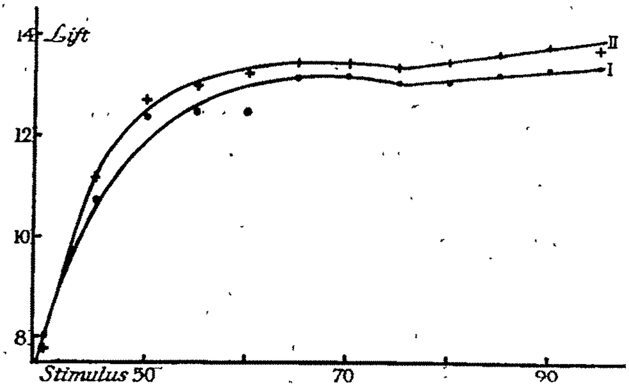

FIG. 67.—Graphic representation of Experiment 20 of Tigerstedt and Willhard. The lift as a function of the amount of the stimulus in arbitrary physical units.

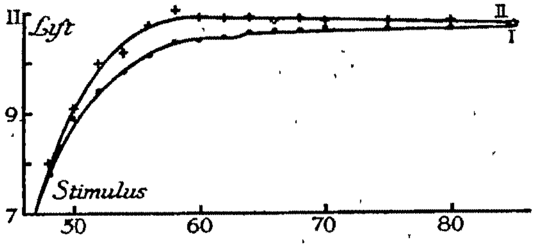

FIG. 68.—Graphic representation of Experiment 28 of Tigerstedt and Willhard. The lift as a function of the amount of the stimulus in arbitary physical units.

diverge towards the end, and that the second curve lies above the first. This proves that the muscle is temporarily deformed by the supramaximal stimuli.

Experiment 28 *of Tigerstedt and Willhard* is of the same kind, and shows the same phenomena (fig. 68). The second curve is remarkable, because during the interval of supramaximal stimuli the lifts at first increase by decreasing stimuli. A better proof that the muscle is deformed by strong stimuli could hardly be given. The lines representing the relation between stimulus and lift for supramaximal stimuli are approximately straight ones, and this harmonizes exactly with the results obtained by Bohr [10] during tetanus of the muscle.

I used the following method to prove the agreement between the formula and the observations. For $R = C_1(P - \Delta) = 0$, the value of the function, or $-l\,[C_1C_2]$, is also zero; this means that the stimulus in physical units, P, will have no visible effect until P surpasses the threshold value Δ. Now I plotted the observed values of $-l$ against the values of P and extrapolated this curve graphically. In this way Δ is fixed. By means of Δ the value of the stimuli $(P - \Delta)$ is determined. Next I tried to determine graphically the top of the tracing. In choosing the experiments, where this was possible, I eliminated those in which the plastic deformation was very dominant. At the top of the curve R is approximately 76, and I pose, therefore, $R_{\text{max.}} = 76$ $C_1\,(P_{\text{max.}} - \Delta)$. $P_{\text{max.}}$ means the value of P in physical units, for which the lift is a maximum. By the determination of the top, $P_{\text{max.}}$ is also fixed, and hence $C_1 = \frac{76}{P_{\text{max.}} - \Delta}$. Because P is given in arbitrary units, the determination of C_1 is always possible. C_1 once fixed in this way, the value of the stimulus $C_1\,(P - \Delta)$ may be determined for the whole scale of stimuli. I should propose to call this method the method of the equivalent stimuli, because it assumes that those stimuli are physiologically equivalent which produce the maximum lift. I will not enter upon a discussion of the intrinsic value of this proposition, because it is only applied here for the sake of convenience to have an easy method of testing the formula. When the reduced stimuli $C_1\,(P - \Delta)$ have been calculated, we may also determine the value of the function

$$-\,l[C_1C_2] = C_1\,(P - \Delta)\,\{\,M \log.\, C_1(P - \Delta) - 1\,\}.$$

The top of the curve agrees with $C_1\,(P - \Delta) = 76$, and at that moment the value of the function is approximately 13·98; hence I pose—

$$L_{\text{max.}} = 13{\cdot}98 = l_{\text{max.}}\,[C_1C_2], \text{ or}$$

$\frac{13{\cdot}98}{l_{\text{max.}}} = C_1C_2$; $l_{\text{max.}}$ means the value of l at the top or the maximum lift. By means of this relation C_2 is calculated. There are other methods to test the agreement between the formula and the observed values of l, which are equally simple, but by the method of the equivalent stimuli the influence of the plastic deformation is for a great deal eliminated.

(I) *Experiments of Tigerstedt and Willhard*, 1883 [44]. — These observers used a muscle-nerve preparation of the frog. The nerve was stimulated by electrical stimuli.

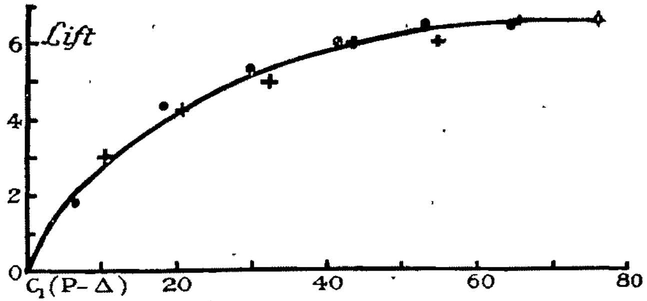

FIG. 69.—Graphic representation of Experiment 6 of Tigerstedt and Willhard. The lift as a function of the reduced stimuli.

Experiment 6.—The nerve is excited by make-induction currents. The mean intensities of the induced currents are proportionate to the numbers given in the second column of Table LII. The stimuli in physical units, P, are therefore proportionate to the mean intensities of the induction currents. Because the mean velocity with which the current increases is approximately independent of the intensity, it is only this last quantity which forms the independent variable of the experiment. The stimulations of the nerve succeeded each other at equal intervals of eight seconds. In the third column the reduced stimuli are given; in the fourth row the lift as observed by Tigerstedt and Willhard; in the fifth column the value of the lift calculated by means of the constants of the sixth column. Fig. 69 is the graphic representation of Table LII. The curve of this figure represents the lifts as a function of the reduced stimuli, calculated by means of the constants of the sixth row. The observed values of the lift are indicated by dots when belonging to a series of increasing stimuli, and by crosses when belonging to a series of decreasing stimuli. In this experiment the two curves calculated by means of the method of the equivalent stimuli coincide.

In order not to burden this part of my research, I will publish the results in as condensed a form as possible. Hence it may suffice to mention only the fact that Experiment 27A of Tigerstedt and Willhard gives the same result without publishing the numerical data. The two curves approximately coincide, and the agreement between the observed and the calculated values of the lift is of the same kind as in the preceding experiment.

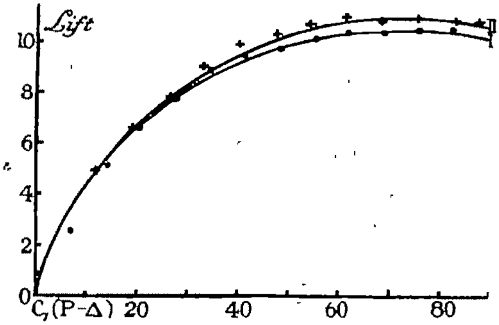

FIG. 70.—Graphic representation of Experiment 28 of Tigerstedt and Willhard. The lift as a function of the reduced stimuli.

Experiment 28 of Tigerstedt and Willhard.—This experiment is of the same nature as the preceding one. The nerve is stimulated by break induction shocks. Table LIII contains the numerical results, and fig. 70 is the graphic representation of the experiment. In this case the two curves do not coincide. The curve marked I, belonging to the series of increasing stimuli, lies below the curve marked II, pertaining to the series of decreasing stimuli. The curves coincide at the beginning and diverge towards the end. This fact proves that a long sequence of increasing stimuli, and especially the stronger and supramaximal stimuli, have temporarily deformed the muscle. This experiment shows also clearly the deviations between the observed and the calculated values of the lift for small values of P.

Experiment 20 of Tigerstedt and Willhard shows the same facts, and the agreement between the observed and the calculated values of the lift is the same.

Experiment 38 of Tigerstedt and Willhard.—In this case the nerve was stimulated by the make and break of a constant current of increasing intensity. Also in this case the mean velocity, with which the current increased or decreased, was approximately constant, independently of the intensity of the constant current. Table LIV and fig. 71 contain the results of the experiment.

Experiment 32A *of Tigerstedt and Willhard* (Table LV, fig. 72).—In this experiment the nerve was curarized and the muscle stimulated directly with break-induction currents. In this case also there exists agreement between the observed and the calculated values of the lift.

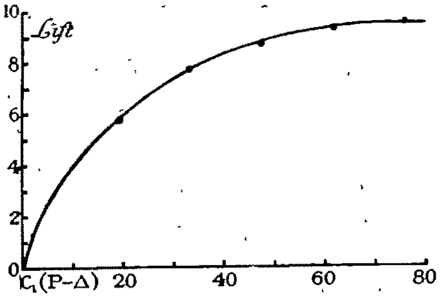

FIG. 71.—Graphic representation of Experiment 38 of Tigerstedt and Willhard. The lift as a function of the reduced stimuli.

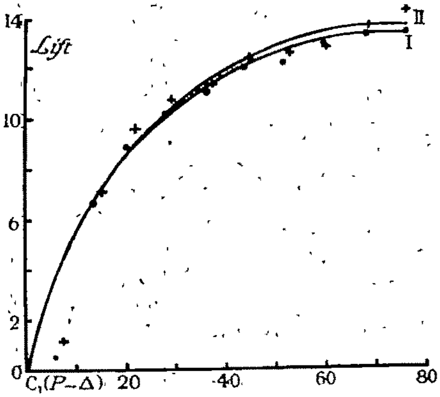

FIG. 72.—Graphic representation of Experiment 32A of Tigerstedt and Willhard. The lift as a function of the reduced stimuli.

The deviation for small values of P is very marked. Experiment 33A of the authors is of the same kind, and yields similar results.

(II) *Experiments of von Kries* [27] 1884.—von Kries worked with the M. gastrocnemius of the frog after destruction of the medullary cord. The nervus ischiadicus is stimulated by a uniformly increasing current (Feder-rheonom). The velocity of the increment of the current was constant throughout and only the intensity varied. The numbers in the second column are proportionate to the intensities of the exciting current. Hence there is only one independent variable in the experiment.

Experiment 2 *of von Kries* (Table LVI, fig. 73).—The nerve was excited by a current which reached its maximum intensity in about 0·05 second.

Experiments 1 *and* 3 give the same result.

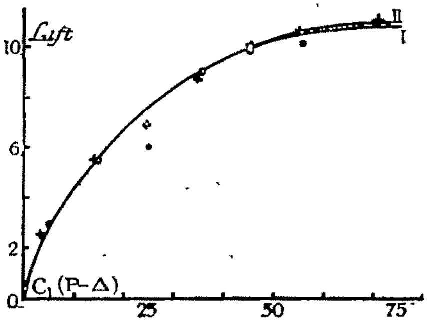

FIG. 73.—Graphic representation of Experiment 2 of von Kries. The lift as a function of the reduced stimuli.

(III) *Experiments of Cybulski and Zanietowski* [16], 1894.—The experiments of the second isotonic series are only suited to my purpose. The authors used the gastrocnemius nerve preparation of the frog. The nerve is stimulated by means of condenser discharges; the capacity of the condenser is 1×10^{-8} F. The charge of the condenser was varied. It may be concluded, from the statements made by the authors about their apparatus, that the mean intensity of the discharge current increases approximately proportionately to the charge of the condenser. The velocity of the increment of the discharge current is but slightly affected by the variation of the mean intensity, so that also in this case there is only one independent variable, which is proportionate to the charge of the condenser. The stimulus in physical units is therefore expressed in coulombs in agreement with the authors.

Experiment 3 *of Cybulski and Zanietowski*, with omission of the first ten observations (Table LVII, fig. 74). This experiment is remarkable for the fact that it shows so very clearly the deformation of the muscle by supramaximal stimuli. The Curve AB represents the relation between the stimulus in physical units and the lift; Curve BC is the continuation of the experiment. It represents the "lifts" for the supramaximal stimuli. It seems evident to me that in a continuously proceeding experiment, the Curve AB cannot represent the same phenomenon as the Curve BC. It is obvious that the Curve BC is not the direct continuation of the Curve AB and that this Curve BC does not approach

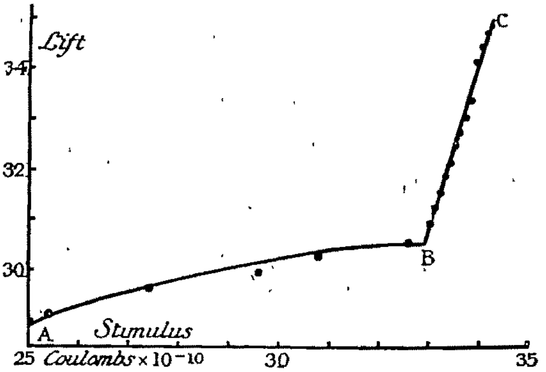

FIG. 74.—Graphic representation of Experiment 8 of Cybulski and Zanietowski. The lift as a function of the amount of the stimulus in arbitrary physical units.

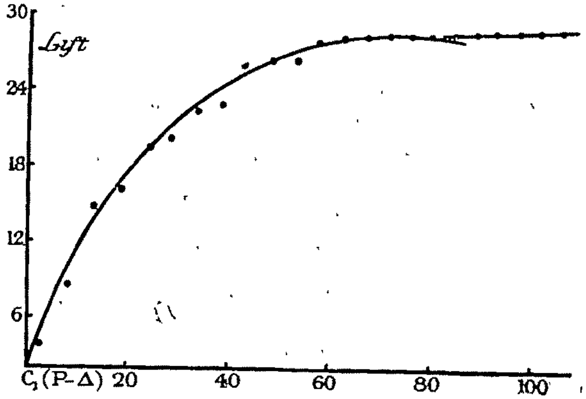

FIG. 75.—Graphic representation of Experiment 2 of Cybulski and Zanietowski. The lift as a function of the reduced stimuli.

asymptotically any limiting value. The Curve BC is rectilinear in the same way as in the experiment of Tigerstedt and Willhard, and represents the deformation of the muscle by supramaximal stimuli.

Experiment 2 of the same authors (Table LVIII, fig. 75) proves the agreement between the observed and the calculated values of the lift. The top of the curve lies in the neighbourhood of P = 31·9, and from that point on the divergence begins. The line uniting the "lifts" caused by the supramaximal stimuli is a straight one also in this case.

Experiments 6A *and* 6B.—Both experiments are made with the same preparation and under the same experimental conditions. Experiment 6A precedes Experiment 6B. The charge of the muscle in Experiment 6A amounted to 5 grm., and in Experiment 6B to 10 grm.; notwithstanding this difference the Curve VIB lies above the Curve VIA. This experiment proves, therefore, again, that the muscle is deformed, more especially by strong stimuli (Table LIX, fig. 76).

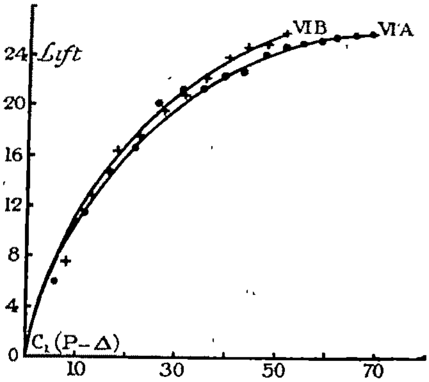

FIG. 76.—Graphic representation of Experiments 6A and 6B, of Cybulski and Zanietowski. The lift as a function of the reduced stimuli.

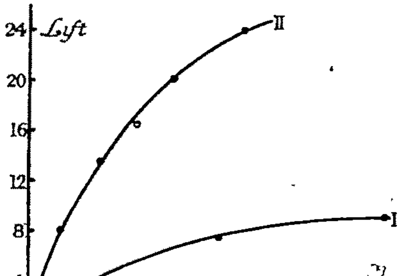

FIG. 77.—Graphic representation of an experiment of Cybulski and Zanietowski. The lift as a function of the reduced stimuli. Curve I twitches, Curve II tetani of short duration.

Experiment 5 of Cybulski and Zanietowski yields the same result.

Finally, I reproduce an experiment where the authors produced alternately a twitch and a tetanus of short duration (Table LX, fig. 77). The discharge current of the condenser excited the nervus ischiadicus of a nerve-muscle preparation of the frog. The tetani were obtained by repeating the discharge at a rate of ·22·5 per second. We see that in the case of tetani of short duration the formula is still applicable, but that the constants are different in the two instances.

Cybulski and Zanietowski also observed the phenomenon that the lift may decrease by increasing stimulus, when exciting the muscle directly, but they attributed it to fatigue (loc. cit., p. 142).

(IV) *Experiment of Waller* [45], 1895.—Waller used the nerve-muscle preparation of the frog. The nerve was excited by induction shocks of increasing strength for periods of one-eighth of a second. The interval between the successive stimuli is one minute. Table LXI contains the data of Waller's experiment, and fig. 78 shows how far the observed and calculated values of the lift agree.

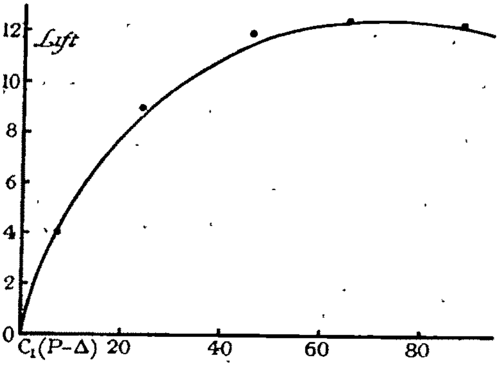

FIG. 78.—Graphic representation of an experiment of Waller. The lift as a function of the reduced stimuli.

(V) *Experiments of Keith Lucas* [30], 1909.—The author used the M. cutaneus dorsi of the frog. The animal was plunged in a bath of Ringer solution. The proprioceptive reflex arc was intact, so that he worked under the same experimental conditions as I did. The nerve on its course to the muscle was stimulated with a series of break-induction currents. The mean intensity of the current is by some indirect way determined by means of a ballistic galvanometer. The strength of the current increased in steps in the successive stimulations. These steps, however, were very small, so that often the passive resistances of the physiological system are not overcome. Hence, two or even three stimuli of increasing strength yield often the same lift.

Experiment 6 *of Keith Lucas* (Table LXII, fig. 79).—"In this experiment the exciting current was not graded finely during the first few observations, so that the steps of the rise are not seen." I choose this experiment exactly for that reason. The experiment shows clearly the deviation at the beginning, and also the existence of a maximum at P = 45·5. Beyond the top the plastic deformation by the supramaximal stimuli is obvious. Experiments 1 and 1A prove also the agreement between the observed and the calculated values of the lift. Especially for the larger values of P the agreement is evident.

(VI) *Experiments of Gildemeister* [20], 1911.—Gildemeister used the discharge current of a condenser, with which he stimulated a nerve-muscle preparation.

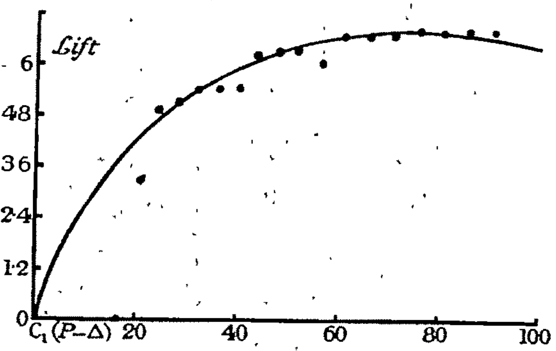

FIG. 79.—Graphic representation of Experiment 6 of Keith Lucas. The lift as a function of the reduced stimuli.

Experiment 11 *of Gildemeister* (Table LXIII, fig. 80).—A nerve-muscle preparation of the frog is used for this experiment. Every observation is repeated twice. The experiment is composed of two sets of observations. The observations of Curve I are obtained by means of quick discharges of the condenser, and those of Curve II are the result of slow discharges. Because the stimuli in the two cases are qualitatively different as the rate of discharge varies, the constants are different in the two sets of observations.

Experiment 5 *of Gildemeister* is of the same kind and yields similar results.

(VII) *Experiments of May* [32] 1911, 1912.—May worked with the tonic M. triceps suræ of the cat. The animal was slightly narcotized with chloroform. The muscle remained in normal relation with the nervous system, so that the proprioceptive reflex arc was intact. The Achilles tendon was cut through, the muscle carefully loosened and attached to the rectilinear myograph of Keith Lucas. A normal salt solution at body temperature flowed through the muscle in order to eliminate the influence of the chloroform upon the muscle. May operated therefore with a normal tonic muscle and under the same conditions as I did. The muscle was stimulated either directly or indirectly through the motor nerve, by a current of 2 milliamperes, interrupted by a Leduc interrupter. The number of interruptions per second was chosen in such a way that each elementary current could display its full physiological effect, and that the strength of each elementary current amounted to but a fraction of a milliampere. The appliance of a Leduc interrupter makes it possible to work with currents of such a feeble intensity, and the increment of the stimulus is obtained by augmenting the frequency of the interruptions. The numbers in the second column of Tables LXIV, LXV, LXVI, denote the number of interruptions per second. The stimulation always lasted one second and the muscle was loaded with 50 or 100 grm. The use of very weak currents is probably the cause that plastic deformation seems to play no rôle in

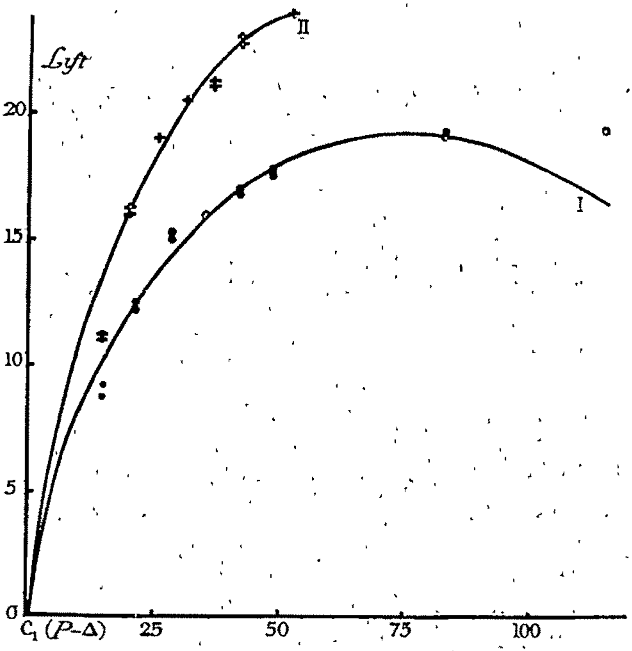

FIG. 80.—Graphic representation of Experiment 11 of Gildemeister. The lift as a function of the reduced stimuli.

these experiments. The absence of any perceptible deformation follows not only from my calculation, but the author himself remarks that "if the frequency instead of being increased is diminished step by step, the tracing obtained is the negative image of those obtained by increasing stimuli."

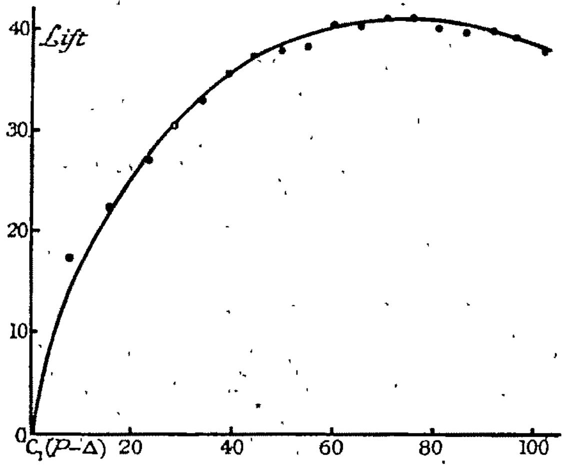

FIG. 81.—Graphic representation of Experiment 2 of May. The lift as a function of the reduced stimuli.

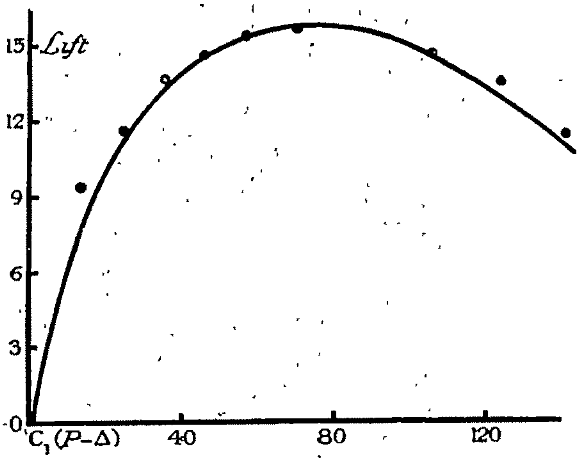

FIG. 82.—Graphic representation of Experiment 3 of May. The lift as a function of the reduced stimuli.

Experiment 2 *of May* (Table XLIV, fig. 81).—The muscle was excited directly by two platinum iridium electrodes, one placed under the top of the muscle, the other near the Achilles tendon. The curve shows obviously a top in the neighbourhood of P = 150 and then decreases. In this case the agreement, especially for the decreasing part of the curve, is evident, because no deformation occurs.

Experiment 3 *of May* (Table LXV, fig. 82).—This experiment is of the same kind as the preceding one. In this case the decrease of the

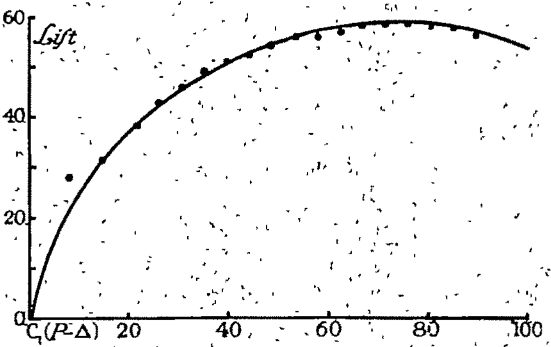

FIG. 83.—Graphic representation of Experiment 4 of May. The lift as a function of the reduced stimuli.

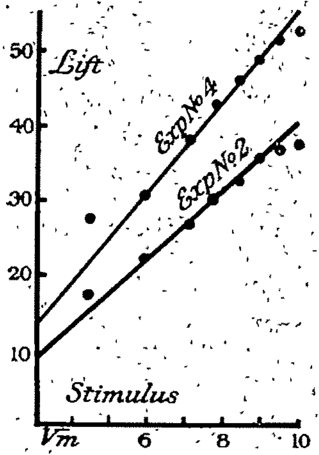

FIG. 84.—Graphic representation of Experiments 2 and 4 of May. The lift as a function of the square root of the number of interruptions (*m*).

lift by increasing stimulus is very considerable. This experiment is therefore remarkable, because it shows the close agreement between the observed and the calculated values of the lift for a considerable part of the descending branch of the curve. This fact is the more remarkable, because the curve is not symmetrical in respect to the maximum ordinate.

Experiment 6 is of the same nature and proves also the close agreement between the observed and the calculated values of the lift.

Experiment 4 *of May* (Table LXVI, fig. 83).—In this experiment the nervus ischiadicus was stimulated with very weak currents. As may be seen from fig. 83, the agreement between the observed and the calculated values of the lift are very close.

Finally, I thought it interesting to test the renowned formula of Nernst [34] on the experiments of May. The formula of Nernst applied in this case leads to the result that the increment of the lift ought to be proportionate to the square root of the number of interruptions. In fig. 84 I plotted these square roots against the lifts. We see, indeed, that the agreement is very close in the beginning, but that the divergence begins in the neighbourhood of the sixth observation. The physical conception of Nernst of the physiological problem is, therefore, confirmed by these experiments, at least for small values of P.

We conclude from all these experiments that the formula

$$-l = \frac{1}{C_1C_2}\ [C_1(P - \Delta)\ \{\ \mathrm{lgn.}\, C_1(P - \Delta) - 1\ \}]$$

really represents the relation between stimulus and lift, inasmuch as a formula which contains only one independent variable may be able to represent this relation. Hence we conclude *that the analysis of the extension curve of the tonic muscle, which leads to the formula* $(-l)$, *is confirmed by the facts.*

More especially we may conclude, in agreement with Wedenski [46], Woolley [48], and others, *that the lift considered as a function of the stimulus in physical units is a maximum phenomenon, and that the lift decreases when the stimuli become supramaximal. This decrement of the lift is in most cases, however, counterbalanced, and therefore hidden, by the plastic deformation of the muscle.*

CHAPTER IV.—TONUS AND TENDON REFLEX.

The analysis of the extension curves of the tonic muscle has led to the result that the tonic muscle is continually in a state of slight contraction, combined with a state of exalted plasticity. The source of the stimuli which maintain this peculiar state of the muscle are the movements of the body which excite continuously the proprioceptors of the whole apparatus of locomotion. The physiological facts brought into connexion with the morphological discoveries of Boeke caused us to conclude that plasticity is the chief property of the sarcoplasmatic part of the muscle and that the maintenance of a slight state of contraction is due to the striped apparatus. Hence one component of the tonus, the plasticity, is controlled by the sympathetic system, and the other component, the contraction, remains under the control of the motor cell of the anterior horn.

From the two components, by which muscle tonus is composed, the component "contraction" is the more variable. For, in the first place, preformed spinal mechanisms may influence the state of contraction of the muscle, and in the second place voluntary impulses may change the state of contraction. The plasticity, on the contrary, as being the autonomic component of the tonus, is withdrawn from our will, and therefore is the more stable component. The clinical definition, which is usually given of muscle tonus, viz., a state of slight contraction, keeps only one component in view, and especially the more variable one. Hence it is obvious that clinical observations, as well as experiments, based upon that definition have given contradictory results, and have failed to prove beyond a doubt the existence of tonus.

It seems desirable to me to have clinical terms to designate the two components of the tonus, and I should be inclined to propose the terms "*contractile tonus*" and *plastic*,[1] or *autonomic*, *tonus*, as perhaps the most adequate ones.[2] Instruments suitable for the measurement of the two different qualities of the tonus separately do not exist at the moment, as far as I know. Hence I will give a rather rough, but in many respects sufficient, clinical method. The M. biceps of the arm may for this purpose serve as an example. We begin to divert the attention of the patient from what we are doing, because attention markedly influences the state of contraction of our muscles. Next we take the wrist with one hand, fix the shoulder with the other and then we bend and extend the arm several times. Finally, we extend the arm moderately and take the belly of the biceps between thumb and fingers of the hand which fixed the shoulder. Now we first try to displace the muscle to and fro across its length. The resistance felt by this movement gives us an impression of the state of contraction of the muscle, or of the contractile tonus; next we compress the belly of the muscle gently between thumb and index and this compression affords us an impression of the plasticity of the muscle, or of the plastic tonus. If we repeat this proof two or three times, we succeed fairly well in getting an idea of the tonic state of the muscle. The repeated movement of bending and extending the arm serves to activate the proprioceptive reflex arc and to eliminate as much as possible the influence of accidental stimuli upon the tonus of the M. biceps. Because the two components of the tonus are independent of each other we may find little resistance by the displacement of the muscle combined with marked plasticity, or the reverse, but in most cases resistance and plasticity go *pari passu* under normal conditions.

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[1] Sherrington (*Quart. Journ. of Exp. Phys.*, 1909, vol. iii) uses also the term "plastic tonus," but in a somewhat different sense.

[2] The investigations on the tonus of smooth muscles have led to similar results. Plasticity, for instance, agrees in its essential features with "Substanztonus" of F. Schultz, and the "neurogenè tonus" of the same author resembles in many respects contractile tonus. This agreement is not accidental, since the saroplasmatic part of the muscle is morphologically and physiologically the analogue of the smooth muscle.

I do not think that it will be easy to construct a simple instrument, suitable for clinical use to measure contractile tonus, but the degree of plastic tonus could perhaps be estimated by means of a set of test-cylinders of rubber of varying plasticity. If we alternately compress the muscle and the test-cylinders, it will be possible to find out the cylinder, which approximately equals the muscle in plasticity.

The tendon phenomena have always been considered as connected with muscle tonus, but about the nature of this connexion opinions are divided. The majority of the observers believe that the tonic state of the muscle is a true reflex action, and the tendon phenomenon a direct muscular response, of which only the tonic muscle is capable (Gowers and others). The chief argument, upon which this supposition is based, is rather a negative one—namely, that the latent period between the tap on the tendon and the contraction is too short for a reflex. Of recent years, however, Sherrington [41], Snijder [42], and others, have found indubitable muscular reflexes with as short a latent period as the tendon phenomenon.

In my opinion, in agreement with the minority of the clinical observers (Snijder and others), *the tendon phenomenon is a true reflex, the typical reflex manifestation of the proprioceptive reflex arc.* The tap on the tendon is the stimulus which elicits reflexly the contraction. The knee-jerk may serve as example for the description of the mechanism of the tendon reflex. If we will elicit a knee-jerk, we begin to stretch the M. quadriceps by bending the knee sufficiently. The tap on the tendon produces under these circumstances a brief and sudden increment of the stress of the muscle. The duration of this variation of the stress, however, is too short to produce a displacement or a lengthening of the muscle as a whole, but it evokes a stress-wave, travelling through the muscle. This wave, which is directly visible, may also be recorded by means of a tambour of Marey. The variation of the tension, provoked by the stress-wave, forms the adequate stimulus for the proprioceptors of the muscle and the tendon. The initial stretching of the muscle, is, therefore, necessary to create the physical condition needed for the production and the propagation of the stress-wave through the muscle. The response of the proprioceptive reflex arc to the stimulation of its proprioceptors is a contraction. The direct observation of the contraction as well as the records on a drum prove that one of the components of the contraction is a single twitch. The electric phenomena, accompanying the contraction, recorded by Wertheim Salomonson [47], and by Dittler and Günther [17], by means of the string galvanometer, are in full harmony with this. A close observation shows, however, that the stadium decrementi of the twitch always seems lengthened. This lengthening is very obvious in the tracings of Gotch [21]. Sometimes the descending branch of the twitch may exhibit a slow secondary elevation (Shepherd Franz [19]). The lengthening or the secondary elevation is caused by a tonic contraction. We conclude from these facts *that the contraction following the tap on the tendon is of a twofold nature, viz., a twitch upon which is superposed a tonic contraction.* That we have really to deal with a superposition of two different forms of contraction is convincingly proved by Graham Brown [22], de Boer [9], and others.

The two components composing the muscular contraction which follows reflexly the tap on the tendon, seem to be independent of each other. This is demonstrated also by an experiment of de Boer on cats. de Boer cut through the rami communicantes of the sympathetic chain, and destroyed in that way the plastic tonus. Notwithstanding the loss of the plastic tonus, the tendon reflex could be elicited and was even brisk. This briskness of the tendon reflex is probably caused by the absence of the tonic contraction. Clinical observation tends to the same conclusion. There are, namely, some forms of locomotor ataxia in which the twitch is diminished or absent, and in these cases the slow and sluggish contraction seems caused only by the tonic component. Also a partial dissociation between the twitch and the tonic contraction occurs.

Shortly summarizing, we may say that the term "tonus" designates the state of the muscle when continually subjected to the influence of weak stimuli, emanating from its own proprioceptors. By the term "tendon reflex" we indicate the sudden variation of the tonus, evoked reflexly by a brief but vigorous stimulus of the proprioceptors of the muscle and the tendon. Hence *tonus and tendon reflex as to their intimate nature are identical,* and the tendon reflex may therefore serve as a touchstone for the tonic state of the muscle. *In the tonus however, the plastic component seems to prevail, and in the tendon reflex the twitch is the most obvious phenomenon. Hence it is evident that the tendon reflex will afford us especially an impression about the contractile component of the tonus.*

It follows from our conception that the tendon phenomenon is a true reflex, and that the muscular response is of a twofold nature, that the terms used to designate the different modalities of the tendon reflex are very imperfect. I will leave aside here the question of the relation between the intensity of the tap on the tendon and the duration of the latent period between the tap and the contraction, as well as the question of the threshold value. What interests us, clinically, is the relation between the intensity of the tap on the tendon and the mechanical effect. This effect may be normal, diminished, or exaggerated, but these terms remain equivocal unless we add, which of the two components is normal, diminished, or exaggerated. Under normal conditions the variation of the two components usually goes *pari passu,* but under pathological conditions this is no more the case. When the twitch prevails, we get the impression that the reflex is brisk, and when the tonic contraction dominates that it is slow. I think, therefore, that the combination of the terms "normal, diminished, exaggerated," with brisk or slow will be sufficient for practical use, though they do not cover the whole field of phenomena.

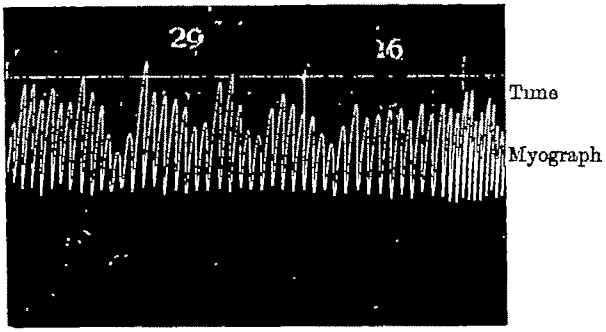

FIG. 85.—Clonus tracing of the tonic M. triceps suræ of the spinal cat. The speed of the drum was not uniform, as may be seen at the time marking.

When the irritability of the reflex centre is exalted, and t reflex exaggerated, the stimulation of the proprioceptors, callec the reflex contraction, suffices to again engender a contractic repetition of the reflex is called "clonus."

Fig. 85 is the reproduction of a clonus tracing of the M. tri of a spinal cat. It is evident from this tracing that clonus is pure twitches, in which no trace of a tonic contraction is to be This is always the case in clonus tracings in man. Fig. sents another clonus curve, also of a spinal cat. At the indicated by the dropping of the signal line, the load attach

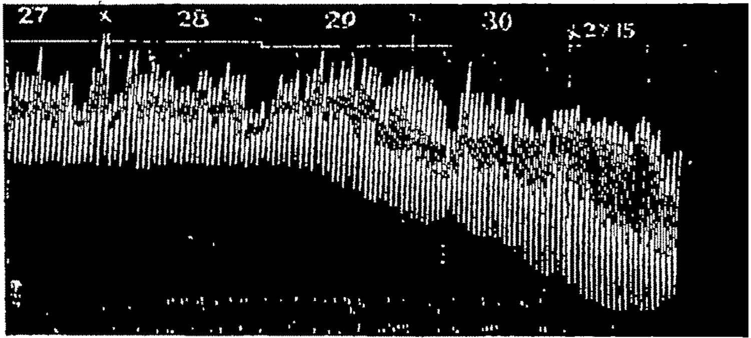

FIG. 86.—Clonus tracing of the tonic M. triceps suræ of the spinal cat. At th indicated by the dropping of the signal line, the load begins to increase unif

tendon of the muscle is uniformly increased. The increa acting as a stimulus for the proprioceptors, accelerates the of the clonus by about 7 per cent., and approximately do excursion of the movement.[1]

Figs. 87 and 88 show what precedes the commenceme clonus. At A the muscle exhibits a tonic shortening, then the the muscle remains constant till B. Again the muscle shorten shortening is accompanied by a clonus. In the case repres fig. 88 the clonus was initiated by a jerk at the tendon [×]. like a tendon-reflex, is in most cases initiated by a jerk or a p tendon, but sometimes (as in fig. 87) the clonus seems pseud neous. In these last cases there are usually movements in an groups of muscles which initiate the clonus of the agonist. from my experiments that the beginning of a clonus is alway panied by a tonic shortening, but that inversely a tonic shortening is not always followed by a clonus. Hence I conclude that the tonic shortening is a necessary factor in the production of a clonus, or *that a clonus is composed by a series of twitches superposed upon a tonic shortening.*

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[1] This fact agrees with the form of the action current as recorded b Salomonson [47].

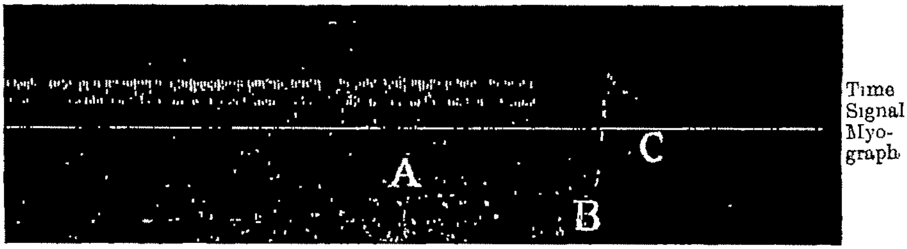

FIG. 87.—Clonus tracing of the tonic M. triceps suræ of the spinal cat. At A and at B a tonic shortening preceding the clonus, at C tonic lengthening of the muscle following the cessation of the clonus..

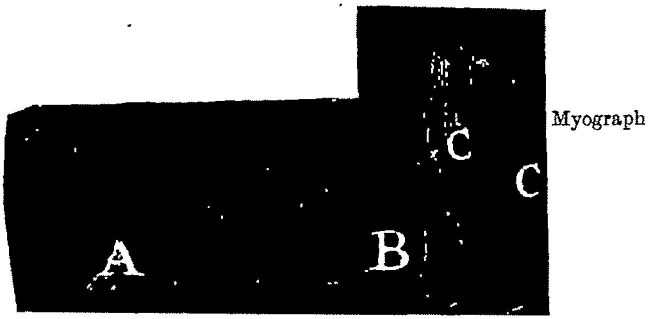

FIG. 88.—Clonus tracing of the tonic M. triceps suræ of the spinal cat. At A and at B a tonic shortening preceding the clonus; at C tonic lengthening of the muscle following the cessation of the clonus; at × the clonus is initiated by a jerk at the tendon.

The cessation of a clonus is always accompanied by a tonic lengthening of the muscle (figs. 87, 88, C). Fig. 89, also a clonus from the triceps suræ of a spinal cat, shows a peculiarity of the tonic lengthening. At the beginning of the experiment, the muscle slowly lengthens, then, at the moment indicated by a cross, I pulled at the tendon, and a clonus set in. Very soon the clonus dies away, and at C the tonic lengthening begins. Now the line representing the tonic lengthening shows clearly the staircase phenomenon. This staircase phenomenon is also visible upon the line of the tonic shortening. This line, however, is in most cases very steep, and this is the reason that the steps are not very obvious. The ascending line of fig. 89 exhibits two steps, each a little below the top of the first and the second twitch. We conclude from these facts *that the tonic shortening of the muscle preceding the clonus is a tonic tetanus, and that this tetanus is the result of the exalted irritability of the proprioceptive reflex arc. A clonus is therefore a series of twitches elicited reflexly, and superposed upon a tonic tetanus.* This result is in harmony with our conception of the duality of the tendon reflex.

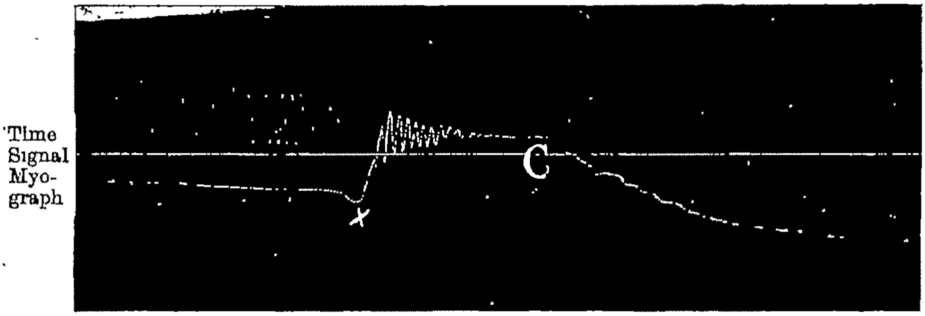

FIG. 89.—Clonus tracing of the tonic M. triceps suræ of the spinal cat. At [×] the clonus is initiated by a pull at the tendon. At C a slow tonic lengthening of the muscle. The line representing the lengthening of the muscle shows the staircase phenomenon.

We arrive, therefore, at the general result that the tonus, the tendon reflex, and the clonus are closely allied phenomena, and that they are composed by an element "contraction," due to the action of the motor cell of the anterior horn and by an element "plasticity," due to the action of a sympathetic motor cell of the cord. *In the tonus the autonomic component prevails; in the tendon reflex the twitch dominates the tonic contraction; in the clonus a series of twitches is superposed upon a tonic tetanus.*

The results at which we have arrived, viz., the morphological and functional duality of the striped muscle, may throw light upon several problems of pharmacology and neuropathology.

My experiments tend, for instance, to the conclusion that several drugs, e.g., strychnine, affect preponderantly the sarcoplasmatic part of the muscle. Fig. 90 is a reproduction of a reflex contraction of the M. gastrocnemius of the frog, elicited by stimulation of the skin of the foot. The twitch does not seem obviously altered, but the height and the duration of the tonic contraction (tC) are strongly increased. The same effect may be produced by cooling down the whole animal.

The cutting through of the brain-stem between thalamus and midbrain (Sherrington [40]) provokes a similar effect. By this section the lower sympathetic centres are divided from the higher centres, situated in the base of the brain. I have only qualitative experience of the state, designated by Sherrington "decerebrate rigidity"; but it seems very probable to me that this rigidity is at least partly due to a spasm of the sarcoplasmatic part of the muscle.[1] Hence one of the components in the decerebrate rigidity is a spasm of sympathetic origin, caused by the prevailing of the cerebellum. This opinion harmonizes with the views of Sherrington [41], who considers the cerebellum as the main ganglion of the proprioceptive system.

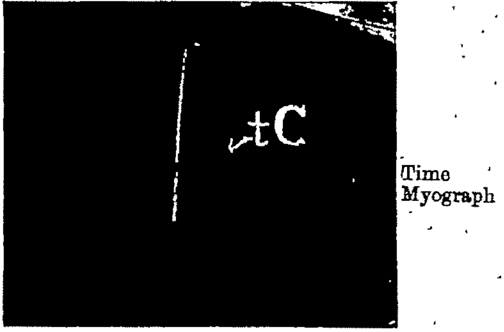

Fig. 90.—Reflex contraction of the tonic M. gastrocnemius of the frog, elicited by stimulation of the skin of the foot, after injection of strychnine. (tO) tonic part of the contraction. The enlargement is about 10 times. The time is marked in tenths of seconds.

Another example produces the myopathic forms of progressive muscular atrophy. The muscle by its sympathetic innervation comes upon the same line with the other internal organs of the body, which all belong to the domain of the sympathetic system. The pathological anatomy of these myopathies make it probable that not only the muscle is diseased, but also a part of the sympathetic system [31]. The primary motor neurone, on the contrary, is always found normal—at least in the beginning of the disease. In this respect the myopathic forms differ from the neurotic forms of muscular atrophy, in which the primary motor neuron is affected.

These few examples may suffice to show that the conception of the duality of the striped muscle may be very fruitful in the future for our knowledge of the nervous system.

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[1] The experiments of Dusser de Barenne tend to the same conclusion (*Folia Neurob.*, 1918, Bd. vii, B. 651).

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APPENDIX.

[THESE TABLES SHOULD BE READ IN CONJUNCTION WITH CHAPTER I.]

(*A*) *The Load Variable.*

TABLE I.—*Experiment* 1 (November 9, 1904).

τ	L	$\frac{\Delta L}{\Delta \tau}$	$\frac{\Delta L}{\Delta \tau} \times \tau$
0.0 × 60	0 0	—	—
$0 + \epsilon$	109·0 (?)	—	—
$\frac{1}{16}$	—	64·0	4 0
$\frac{1}{8}$	117·0	—	—
$\frac{3}{16}$	—	20 0	3·75
$\frac{1}{4}$	119·5	—	—
$\frac{3}{8}$	—	11·0	4·12
$\frac{1}{2}$	122·5	—	—
$\frac{3}{4}$	—	5 0	3·75
1	124 75	—	—
$1\frac{1}{2}$	—	2 75	4·12
2	127·5	—	—
$2\frac{1}{2}$	—	1·5	3·75
3	129·0	—	—
4	—	1·0	4·0
5	131·0	—	—
6	—	0·625	3·75
7	132·25	—	—
$8\frac{1}{2}$	—	0 50	4·25
10	133·75	—	—
$12\frac{3}{4}$	—	0·32	4·0
$15\frac{1}{2}$	135·5	—	—
18	—	0·20	3 6
$20\frac{1}{2}$	136·5	—	—
23	—	0·20	4·6
$25\frac{1}{2}$	137·5	—	—
28	—	0·15	4·2
$30\frac{1}{2}$	138·25	—	—
$35\frac{1}{2}$	—	0·125	4·4
$40\frac{1}{2}$	139·5	—	—
46	—	0·09	4·0
$51\frac{1}{2}$	140·5	—	—
$51\frac{3}{4}$ × 60	—	—	—
		a.m.	4·02
$0 = 51\frac{3}{4}$ × 60	140 5	—	—
$51\frac{3}{4} + \epsilon$	82·25 (?)	—	—
$51\frac{13}{16}$	—	56·0	3·5
$51\frac{7}{8}$	75·25	—	—
$51\frac{15}{16}$	—	18·0	3·4
52	73·0	—	—
$52\frac{1}{8}$	—	10·0	3·75
$52\frac{1}{4}$	70·5	—	—

TABLE I.—*continued.*

T	L	$\frac{\Delta L}{\Delta T}$	$\frac{\Delta L}{\Delta T} \times T$
52⅜	—	5·0	8·12
52½	69·25	—	—
52¾	—	3·5	8·5
53	67·5	—	—
53½	—	2·0	8·5
54	65·5	—	∧∧∧∧∧
54½	—	0·75 (?)	—
55	64·75	—	—
55½	—	1·0	8·75
56	63·75	—	—
56⅞	—	0·71	8·6
57¾	62·5	—	—
59¾	—	0·44	8·5
61¼	60·75	—	—
64½	—	0·25	8·12
66¾	59·5	—	—
69¼	—	0·2	8·5
71¾	58·5	—	—
76¾	—	0·15	8·75
81¾	57·0	—	—
86¾	—	0·1	8·5
91¼	56·0	—	—
94¼	—	—	—
96¾ × 60	55·5^{+}	<0·1 >0·05	<4·25 >2·1
			a.m. 8·5

TABLE II.—*Experiment* 2 (November 7, 1904).

T	L	$\frac{\Delta L}{\Delta T}$	$\frac{\Delta L}{\Delta T} \times T$
0·0 × 60	0·0	—	—
0 +	121·5 (?)	—	—
$\frac{1}{16}$	—	96·0	6·0
⅛	133·5^{+}	—	—
$\frac{3}{16}$	—	32·0	6·0
¼	137·5^{+}	—	—
⅜	—	16·0	6·0
½	141·5^{+}	—	—
¾	—	8·0	6·0
1	145·5^{+}	—	—
1½	—	4·0	6·0
2	149·5^{+}	—	—
2½	—	2·5	6·25
3	152·0	—	—
4	—	1·5	6·0
5	155·0	—	—
7½	—	0·8	6·0
10	159·0	—	—
12½	—	0·5	6·25
15	161·5^{-}	—	—
17½	—	0·35	6·12
20	163·0^{+}	—	—
22½	—	0·3	6·75

TABL[illegible]tinued.

τ		ΔL	$\frac{\Delta L}{\Delta \tau} \times \tau$
25			—
27½			6 85
30	166·0	—	—
32½	—	0 2	6 5
35	167·0	—	∧∧∧∧∧
37½	—	0·25 (?)	—
40	168·25	—	—
42½	—	0·1 (?)	—
45	168 75	—	—
47½	—	0·15	6 6
50	169·5	—	∧∧∧∧∧
55	—	0 15 (?)	—
60	171·0	—	—
65½	—	0·09	6 0
71 × 60	172 0	—	—
		a.m.	**6·22**
0=71 × 60	172·0	—	—
71 + ϵ	104·5 (?)	—	—
71$\frac{1}{16}$	—	128·0	8·0
71⅛	88·5	—	—
71$\frac{3}{16}$	—	40·0	7·5
71¼	83·5	—	—
71⅜	—	21·0	7 9
71½	78 25	—	—
71⅝	—	18 0	8 1
71¾	75·0	—	—
71⅞	—	10·0	8 7
72	72·5	—	—
72¼	—	5·0	7 5
72½	69·0	—	—
72¾	—	4·0	7 0
73	67 0	—	—
73½	—	3·5	8 7
74	63 5	—	—
74½	—	2·5	8 7
75	61 0	—	—
75½	—	1·5	7 0
76	59·5	—	—
77	—	1·25	7 5
78	57·0	—	—
79	—	1 0	8·0
80	55·0	—	—
82½	—	0·7	8 0
85	51·5	—	—
87½	—	0 5	8·2
90	49 0	—	—
100 Extrapolation	43·7	—	—
200 Extrapolation	34 0	—	—
300 Extrapolation	30 0	—	—
400 Extrapolation	27 3	—	—
		a.m.	**7·91**

TABLE III.—*Experiment* 3 (November 15, 1904).

τ	L	$\frac{\Delta L}{\Delta \tau}$	$\frac{\Delta L}{\Delta \tau} \times \tau$
00 0 × 60	0·0	—	—
0 + ϵ	108·5 (?)	—	—
$\frac{1}{8}$	—	86·0	4·5
$\frac{1}{4}$	117·5	—	—
$\frac{3}{8}$	—	12·0	4·5
$\frac{1}{2}$	120·5	—	—
$\frac{3}{4}$	—	7·0	5·0
1	123·5^{-}	—	—
$1\frac{1}{2}$	—	3·0	4·5
2	126·5^{-}	—	—
3	—	1·5	4·5
4	129·5	—	—
6	—	0·75	4·5
8	132·5	—	—
12	—	0·375	4·5
16	135·5^{+}	—	—
24	—	0·19	4·5
32	138·5^{-}	—	—
40	—	0·125	5·0
48	140·5^{+}	—	—
56	—	0·125 (?)	—
64	142·5	—	—
72	—	0·063	4·5
80	143·5^{+}	—	—
88	—	0·05	4·9
96	144·5^{-}	—	—
101 × 60	144·5^{+}	—	—
			a.m. **4·63**
0=101 × 60	144·5^{+}	—	—
101 + ϵ	71·0 (?)	—	—
$101\frac{1}{8}$	—	28·0	3·5
$101\frac{1}{4}$	64·0^{+}	—	—
$101\frac{3}{8}$	—	9·0	3·4
$101\frac{1}{2}$	62·0^{-}	—	—
$101\frac{3}{4}$	—	4·0	3·0
102	60·0^{-}	—	—
$102\frac{1}{2}$	—	2·0	3·0
103	58·0^{-}	—	—
105	—	0·84	3·75
107	54·25	—	—
110	—	0·42	3·75
113	51·75	—	—
117	—	0·22	3·5
121	50·0	—	—
125	—	0·125	3·0
129	49·0	—	—
			a.m. **3·36**

TABLE IV.—*Experiment* 4 (November 15, 1904).

τ	P	Δ P per 60 sec.	L
0·0 × 60	13·00	+ 2·30	0·0
3·0	19·89		14·6
6·0	26·78		29·5
7·5	28·50	− 2·19	28·5
9·0	20·22	+ 2·21	26·0
12·0	26·85		34·0
15·0	33·46		42·0
16·5	30·01	− 2·32	41·0
18·0	26·52	+ 2·12	39·5
21·0	32·89		44·0
24·0	39·26		49·5
25·5	35·06	− 2·40	49·0
27·0	32·06	+ 2·07	47·0
30·0	38·27		50·5
33·0	44·49		55·5
34·5	40·85	− 2·43	55·0
36·0	37·21	+ 2·04	53·0
39·0	43·32		56·5
42·0	49·43		61·5
43·5	45·72	− 2·47	61·0
45·0	42·01	+ 2·00	59·0
48·0	48·02		61·75
51·0	54·03		65·5
52·5	50·31	− 2·48	65·0
54·0	46·58	+ 1·99	64·0
57·0	52·54		66·0
60·0	58·50		70·0
61·5	54·77	− 2·49	69·5
63·0	51·04	+ 1·99	68·0
66·0	57·00		70·5
69·0	62·97		74·0
70·5	59·24	− 2·49	73·5
72·0	55·50	+ 1·98	72·0
75·0	61·44		74·0
78·0	67·39		77·5
79·5	63·66	− 2·49	77·0
81·0	59·92	+ 1·98	76·0
84·0	67·42		78·75
87·0 × 60	71·81		81·0

TABLE V.—*Experiment* 5 (November 16, 1904).

τ	P	Δ P per 60 sec.	L
0·0 × 60	13·45	+ 3·05	0·0
1·5	18·02		8·5
3·0	22·06		19·0
4·5	19·42	− 2·12	16·0
6·0	16·23	+ 3·04	11·0
7·5	20·79		18·0
9·0	25·35		26·0
10·5	22·15	− 2·13	24·5
12·0	18·95	+ 3·02	20·75
13·5	23·48		26·0
15·0	28·02		33·0
16·5	24·79	− 2·16	31·0
18·0	21·55	+ 3·00	27·0
19·5	26·05		32·0
21·0	30·55		39·0
22·5	27·29	− 2·18	36·75
24·0	24·02	+ 2·98	33·0
25·5	28·49		38·0
27·0	32·97		44·0
28·5	29·69	− 2·19	42·5
30·0	26·40	+ 2·97	39·0
31·5	30·85		43·0
33·0	35·31		49·5
34·5	32·00	− 2·21	48·0
36·0	28·68	+ 2·95	45·0
37·5	33·10		48·5
39·0	37·53		53·5
40·5	34·20	− 2·22	52·2
42·0	30·86	+ 2·94	50·0
43·5	35·27		53·0
45·0	39·67		57·0
46·5	36·32	− 2·24	56·5
48·0	32·96	+ 2·93	54·5
49·5	37·35		57·0
51·0	41·74		60·75
52·5	38·37	− 2·25	60·0
54·0	35·00	+ 2·92	58·75
55·5	39·37		61·0
57·0	43·75		64·25
58·5	40·37	− 2·25	63·5
60·0	36·99	+ 2·91	62·0
61·5	41·35		64·5
63·0	45·71		67·75
64·5	42·32	− 2·26	67·0
66·0	38·93	+ 2·90	65·75
67·5	43·28		67·5
69·0	47·64		71·0
70·5	44·24	− 2·27	70·25
72·0	40·84	+ 2·89	68·75
73·5	45·18		71·0
75·0	49·52		74·0
76·5	46·10	− 2·28	73·5
78·0	42·68	+ 2·88	72·0
79·5	47·00		74·0
81·0	51·33		77·5
82·5	47·89	− 2·29	76·25
84·0	44·45	+ 2·87	75·0
85·5	48·76		76·5
87·0	53·06		79·0
88·5	49·59	− 2·32	78·25
90·0	46·11	+ 2·87	77·25
91·5	50·42		78·5
93·0	54·72		81·0
94·5	51·28	− 2·29	80·5
96·0	47·84	+ 2·89	79·75
97·5	52·17		81·0
99·0	56·50		83·25
100·5	53·10	− 2·27	83·0
102·0	49·70	+ 2·91	82·25
103·5	54·07		83·5
105·0	58·44		86·0
106·5	55·07	− 2·25	85·5
108·0	51·69	+ 2·93	85·0
109·5	56·09		86·0
111·0	60·49		89·5
112·5	57·19	− 2·20	89·25
114·0	53·89	+ 3·00	88·0
115·5	58·39		90·0
117·0	62·89		92·5
118·5	59·67	− 2·15	92·25
120·0	56·44	+ 3·07	91·25
121·5	61·04		93·0
123·0	65·64		96·0

TABLE VI.—*Experiment* 6 (December 30, 1904).

P	τ	L	$\frac{\Delta L}{\Delta \tau}$	$\frac{\Delta L}{\Delta \tau} \times \tau$
4·8	0·0 × 60	0 0	—	—
7·8	1	—	—	—
14·8	2	—	—	—
24·8	3	—	—	—
44 8	4	—	—	—
—	5	116.0	—	—
74 8	$0 = 5\frac{1}{10}$	132·0 (?)	—	—
—	$5\frac{7}{40}$	—	21·5	7 0
—	$5\frac{1}{4}$	146 0	—	—
—	6	—	8 0	7 2
—	$6\frac{1}{4}$	150·0	—	—
—	$6\frac{3}{4}$	—	4·25	7·0
—	$7\frac{1}{4}$	154·25	—	—
—	$7\frac{3}{4}$	—	2 75	7·3
—	$8\frac{1}{4}$	157 0	—	—
—	$8\frac{7}{8}$	—	2·0	7·0
—	$9\frac{1}{2}$	159·5	—	—
—	$10\frac{1}{2}$	—	1 37	7·1
—	$11\frac{1}{2}$	162 25	—	—
—	13	—	0·91	7,3
—	$14\frac{1}{2}$	165·0	—	—
—	$16\frac{1}{4}$	—	0·64	7·2
—	18	167·25	—	—
—	20	—	0 5	7·5
—	22	169·25	—	—
—	$23\frac{1}{2}$	—	0 41	7 5
—	25	170 5	—	—
—	$27\frac{1}{2}$	—	<0 35 >0 30	<7.9 >6 7
—	30	$172\ 0^{+}$	—	—
—	$32\frac{1}{2}$	—	<0 30 >0 25	<8·2 >6 9
—	35	$173{\cdot}5^{-}$	—	—
—	$37\frac{1}{2}$	—	<0 25 >0 20	<8·1 >6·5
—	40	174 5	—	—
—	45	—	0 175	7 0
—	50	176 25	—	—
—	55	—	0 15	7·5
—	60	$177{\cdot}75^{+}$	—	—
—	61	$177{\cdot}75^{+}$	—	—
				a.m. **7·27**
44 8	$0 = 61\frac{1}{10}$	166·75 (?)	—	—
—	$61\frac{7}{40}$	—	3·75	0·375
—	$61\frac{1}{4}$	166 0	—	—
—	$61\frac{1}{2}$	—	1 0	0·45
—	$61\frac{3}{4}$	165·5	—	—
—	$62\frac{3}{8}$	—	0·2	0·38
—	63	165·25	—	—
—	64	—	0·125	0 375
—	65	165 0	—	—
—	$67\frac{1}{2}$	—	0 05	0·38
—	70	164 75	—	—
—	75	—	0 025	0 35
—	80	164·5	—	—
—	85	—	<0 025 >0·0125	<0 6 >0·3
—	90	$164\ 25^{+}$	—	—
—	$91\frac{3}{4}$	$164\ 25^{+}$	—	—
				a.m. **0·39**

TABLE VI.—*continued.*

P	τ	L	$\frac{\Delta L}{\Delta \tau}$	$\frac{\Delta L}{\Delta \tau} \times \tau$
24·8	$0 = 91\frac{9}{10}$	153·75 (?)	—	—
—	$91\frac{19}{20}$	—	8·0	0·75
—	92	152·25	—	—
—	$92\frac{3}{8}$	—	1·66	0·71
—	$92\frac{3}{4}$	151·0	—	—
—	$93\frac{1}{2}$		0 5	0 77
—	$94\frac{1}{4}$	150·25	—	—
—	$97\frac{1}{8}$	—	0·13	0·67
—	100	149·5	—	—
—	105	—	0·05	0 65
—	110	149·0	—	—
—	115	—	$<0·036$ $>0·025$	$<0 86$ $>0·58$
—	120	148 75	—	—
—	$121\frac{1}{4}$	148 75	—	—
			a.m.	**0·71**
14·8	$0 = 121\frac{9}{10}$	139·0 (?)	—	—
—	$121\frac{19}{20}$	—	20·0	1·0
—	122	137·0	—	—
—	$122\frac{1}{8}$	—	4	0·9
—	$122\frac{1}{4}$	136 0	—	—
—	$122\frac{1}{2}$	—	1·5	0·9
—	$122\frac{3}{4}$	135 25	—	—
—	$123\frac{1}{8}$	—	0 66	1·0
—	$123\frac{1}{2}$	134·75	—	—
—	$124\frac{1}{2}$	—	0·37	0 98
—	$125\frac{1}{2}$	134 0	—	—
—	$127\frac{3}{4}$	—	0·17	0·98
—	130	133·25	—	—
—	135	—	0 075	0·97
—	140	132·5	—	—
—	146	—	0·042	1·0
—	152	132·0	—	—
			a.m.	**0·97**
7·8	$0 = 152\frac{1}{4}$	120·75 (?)	—	—
—	$152\frac{3}{8}$	—	13 0	1·62
—	$152\frac{1}{2}$	117·5	—	—
—	$152\frac{3}{4}$	—	3·0	1·5
—	153	116·0	—	—
—	$153\frac{1}{2}$	—	1·25	1·56
—	154	114 75	—	—
—	157	—	0·33	1·58
—	160	112·75	—	—
—	165	—	0·125	1 56
—	170	111·5	—	—
—	175	—	0 075	1·7
—	180	110 75	—	—
—	181	110 75	—	—
			a.m.	**1·59**
4·8	$0 = 181\frac{1}{2}$	103 0	—	—
—	$181\frac{5}{8}$	—	10 0	1·25
—	$181\frac{3}{4}$	100·5	—	—
—	182	—	2·5	—
—	$182\frac{1}{4}$	99·25	—	1·25
—	$182\frac{3}{4}$	—	1·0	—
—	$183\frac{1}{4}$	98·25	—	1·25
—	$184\frac{1}{4}$	—	0·5	—
—	$185\frac{1}{4} \times 60$	97·25	—	1·12
			a.m.	**1·22**

TABLE VII.—*Experiment* 6 (December 30, 1904).

P	ΔP	$\frac{\Delta L}{\Delta \tau} \times \tau$	$\frac{\frac{\Delta L}{\Delta \tau} \times \tau}{\Delta P}$
74·8	—	—	—
—	30·0	—	—
44·8	—	0·39	0·013
—	20·0	—	—
24·8	—	0·71	0·036
—	10·0	—	—
14·8	—	0·97	0·097
—	7·0	—	—
7·8	—	1·59	0·230
—	3·0	—	—
4·8	—	1·23	0·41

TABLE VIII.—*Experiment* 7 (January 5, 1905).

P	τ	L	$\frac{\Delta L}{\Delta \tau}$	$\frac{\Delta L}{\Delta \tau} \times \tau$
4·8	0·0 × 60	0·0	—	—
7·8	0·0 + ϵ	14·0 (?)	—	—
—	⅛	—	12·0	1·5
—	¼	17·0	—	—
—	½	—	3·0	1·5
—	¾	18·5	—	—
—	1	—	1·5	1·5
—	1¼	19·25	—	—
—	2	—	0·83	1·66
—	2¾	$20·5^-$	—	—
—	3¼	—	0·5	1·62
—	3¾	$20·75^+$	—	—
—	4¾	—	<0·37 >0·25	<1·78 >1·2
—	5¾	21·5	—	—
—	7¼	—	<0·25 >0·17	<1·8 >1·2
—	8¾	$22·0^+$	—	—
—	11¾	—	<0·17 >0·12	<1·96 >1·47
—	14¾	23·0	—	—
			a.m.	**1·56**
14·8	0 = 15½	41·5 (?)	—	—
—	15¾	—	8·0	2·0
—	16	45·5	—	—
—	16¼	—	3·0	2·25
—	16½	47·0	—	—
—	16⅞	—	1·33	1·83
—	17¼	48·0	—	—
—	18¼	—	0·75	2·1
—	19¼	49·5	—	—
—	20	—	<0·5 >0·33	<2·25 >1·5
—	20¾	$50·0^+$	—	—
—	25¾	—	0·25 (?)	∧∧∧∧∧
—	30¾	$53·0^-$	—	—
			a.m.	**1·99**
24·8	0 = 30⅞	62·0 (?)	—	—
—	31$\frac{1}{16}$	—	8·0	1·5
—	31¼	65·0	—	—

TABLE VIII.—*continued.*

P	τ	L	$\frac{\Delta L}{\Delta \tau}$	$\frac{\Delta L}{\Delta \tau} \times \tau$
—	31$\frac{7}{16}$	—	2·66	1·5
—	31$\frac{5}{8}$	66·0	—	—
—	32$\frac{7}{16}$	—	0·92	1·42
—	33$\frac{1}{4}$	67·5	—	—
—	33$\frac{3}{4}$	—	0·5	1·44
—	34$\frac{1}{4}$	68·0	—	—
—	36$\frac{1}{2}$	—	0·28	1·53
—	38$\frac{3}{4}$	69·25	—	—
—	41$\frac{3}{4}$	—	0·16	1·75
—	44$\frac{3}{4}$	70·25	—	—
			a.m.	**1·52**
44·8	0 = 45$\frac{3}{4}$	90·0 (?)	—	2·62
—	45$\frac{7}{8}$	—	21·0	—
—	46	95·25	—	2·25
—	46$\frac{1}{8}$	—	6·0	—
—	46$\frac{1}{4}$	96·75	—	—
—	46$\frac{1}{2}$	—	3·0	2·25
—	46$\frac{3}{4}$	98·25	—	—
—	47	—	2·0	—
—	47$\frac{1}{4}$	99·25	—	2·5
—	48$\frac{3}{4}$	—	0·75	2·25
—	50$\frac{1}{4}$	101·5	—	—
—	52$\frac{3}{4}$	—	0·35	2·45
—	55$\frac{1}{4}$	103·25	—	—
—	57$\frac{3}{4}$	—	0·45 (?)	ʌʌʌʌʌ
—	60$\frac{1}{4}$	105·5	—	—
			a.m.	**2·39**
74·8	0 = 60$\frac{2}{5}$	127·5 (?)	—	—
—	60$\frac{23}{40}$	—	12·9	2·25
—	60$\frac{3}{4}$	132·0	—	—
—	61	—	3·5	2·63
—	61$\frac{1}{4}$	133·75	—	—
—	61$\frac{3}{4}$	—	2·0	3·0
—	62$\frac{1}{4}$	135·75	—	—
—	63$\frac{3}{4}$	—	0·92	3·0
—	65$\frac{1}{4}$	138·5	—	—
—	67$\frac{3}{4}$	—	0·5	3·75
—	70$\frac{1}{4}$	141·0	—	—
—	72$\frac{3}{4}$	—	0·35	4·4
—	75$\frac{1}{4}$	142·75	—	—
—	80$\frac{1}{4}$	—	0·275	5·5
—	85$\frac{1}{4}$	145·5	—	—
—	90$\frac{1}{4}$	—	0·225	6·75
—	95$\frac{1}{4}$	147·75	—	—
—	100$\frac{1}{4}$	—	0·175	7·0
—	105$\frac{1}{4}$	149·5	—	—
—	110$\frac{1}{4}$	—	0·175	8·75
—	115$\frac{1}{4}$	151·25	—	—
—	120$\frac{1}{4}$	—	0·15	9·0
—	125$\frac{1}{4}$	152·75	—	—
—	130$\frac{1}{4}$	—	0·025 (?)	—
—	135$\frac{1}{4}$	153·0	—	(?)
—	137$\frac{1}{4}$	153·0	—	—
44·8	0 = 137$\frac{5}{8}$	135·5 (?)	—	—
—	137$\frac{3}{4}$	—	6·0	0·75
—	137$\frac{7}{8}$	134·0	—	—
—	138	—	2·0	0·75
—	138$\frac{1}{8}$	133·5	—	—
—	138$\frac{15}{16}$	—	0·46	0·75
—	139$\frac{3}{4}$	132·75	—	—
—	141$\frac{1}{2}$	—	$\begin{cases} <0·21 \\ >0·14 \end{cases}$	<0·86 >0·57

TABLE VIII.—*continued.*

P	τ	L	$\frac{\Delta L}{\Delta \tau}$	$\frac{\Delta L}{\Delta \tau} \times \tau$
—	143¼	$132{\cdot}0^{+}$	—	—
—	147¾	—	0 088	0·83
—	152¼	$131{\cdot}5^{+}$	—	—
			a.m.	**0·75**
24·8	0=153	110·5 (?)	—	—
—	153⅛	—	8·0	1·0
—	153¼	108·5	—	—
—	153½	—	2 0	1·0
—	153¾	107·5	—	—
—	154½	—	0·67	1·0
—	155¼	106 5	—	—
—	156¼	—	0 33	1·25
—	158¼	105·5	—	—
—	160¼	—	0·125	0·88
—	162¼	105·0	—	—
—	164¼	—	0·1	1·2
—	167¼	104 5	—	—
			a.m.	**1·08**
14·8	0=168⅛	89 0 (?)	—	—
—	168¼	—	1·0	1·25
—	168⅜	86·5	—	—
—	168⅝	—	2·5	1·25
—	168⅞	85·25	—	—
—	$169\frac{1}{16}$	—	0·86	1·0
—	169¾	84·5	—	—
—	170½	—	0·5	1·2
—	171¼	83·75	—	—
—	173¼	—	0 25	1·25
—	175¼	82·75	—	—
—	177¼	—	0·125	1·13
—	179¼	82·25	—	—
—	181	—	<0 16 >0·08	<1·8 >0·91
—	182¾	82 0	—	—
			a.m.	**1·22**
7·8	0=183⅛	68·25 (?)	—	—
—	183¼	—	14·0	1·75
—	183⅜	64·75	—	—
—	183¾	—	3·5	1·75
—	184	63·0	—	—
—	184⅜	61·5	1·2	1·65
—	185¼	61·5	—	—
—	187¼	—	0·4	1·8
—	190¼	59·5	—	—
—	193¾	—	0·32 (?)	(?)
—	197¼	57·25	—	—
			a.m.	**1·74**
4 8	0=198⅛	53·0	—	—
—	198¼	—	16·0	2·0
—	198⅜	49·0	—	—
—	198⅝	—	4·0	2·0
—	198⅞	47·0	—	—
—	$199\frac{7}{16}$	—	1 55	2·04
—	200	45·25	—	—
—	201½	—	0·58	1·97
—	203 × 60	43·5	—	—
			a.m.	**2·00**

TABLE IX.—*Experiment* 7 (January 5, 1905)

P	ΔP	$\frac{\Delta L}{\Delta \tau} \times \tau$	$\frac{\frac{\Delta L}{\Delta \tau} \times \tau}{\Delta P}$
4·8	—	—	—
—	3·0	—	—
7·8	—	1·56	0·52
—	7·0	—	—
14·8	—	1·99	0·28
—	10·0	—	—
24·8	—	1·52	0·15
—	20·0	—	—
44·8	—	2·39	0·12
—	30·0	—	—
74·8	—	2·44	0·081
—	30·0	—	—
44·8	—	0·75	0·025
—	20·0	—	—
24·8	—	1·05	0·052
—	10·0	—	—
14·8	—	1·22	0·12
—	7·0	—	—
7·8	—	1·74	0·25
—	3·0	—	—
4·8	—	2·00	0·67

TABLE X.—*Experiment* 8 (November 30, 1904).

P	τ	L	$\frac{\Delta L}{\Delta \tau}$	$\frac{\Delta L}{\Delta \tau} \times \tau$
3·65	0·0 × 60	0·0	—	—
13·65	0·0 + ε	38·5 (?)	—	—
—	⅛	—	32·0	4·0
—	¼	46·5	—	—
—	⅜	—	10·0	3·8
—	½	49·0	—	—
—	¾	—	5·0	3·8
—	1	51·5	—	—
—	1½	—	3·0	4·5
—	2	54·5	—	—
—	2½	—	1·5	3·8
—	3	56·0	—	—
—	4	—	1·0	4·0
—	5	58·0	—	—
—	6	—	0·75	4·5
—	7	59·5	—	—
—	8½	—	0·5	4·25
—	10	61·0+	—	—
—	11	—	1·4 (?)	^^^^^
—	12	64·0	—	—
—	13½	—	0·38	4·5
—	15	65·0	—	—
—	17¼	—	0·22	4·0
—	19½	66·0	—	—
	^^^^^		a.m.	**4·12**
13·65	0 = 20½	63·5	—	—
—	21¼	—	1·33	1·0
—	22	65·5	—	—
—	23	—	0·5	1·25
—	24	66·5	—	—
—	27	—	0·17	1·1
—	30	67·5	—	—
—	34¾	—	0·1	1·4
—	39½	68·5	—	—
			a.m.	**1·19**

TABLE X.—*continued.*

P	τ	L	$\frac{\Delta L}{\Delta \tau}$	$\frac{\Delta L}{\Delta \tau} \times \tau$
	∧∧∧∧∧			
13·65	0 = 40¼	64·5	—	—
—	40⅜	—	6·0	0·75
—	40½	66·0	—	—
—	40¾	—	1·5	0·75
—	41	66·75	—	—
—	42	—	0·37	0·66
—	43	67·5	—	—
—	48	—	0·1	0·72
—	53	68·5	—	—
—	57¼	—	<0·06 >0·03	<1·0 >0·5
—	61½	69·0	—	—
—	64	—	—	—
—	66½	70·0	0·2 (?)	∧∧∧∧∧
			a.m.	**0·75**
	∧∧∧∧∧			
13·65	0 = 67⅓	71·5	—	—
—	68¼	—	0·52	0·6
—	70	72·5^{+}	—	—
—	75	—	0·1	0·7
—	80	73·5	—	—
—	85	—	<0·05 >0·025	<0·85 >0·43
—	90	74·0	—	—
			a.m.	**0·65**
	∧∧∧∧∧			
13·65	0 = 91¾	73·0^{+}	—	—
—	91⅞	—	4·0	0·5
—	92	74·0	—	—
—	92½	—	0·5	0·4
—	93	74·5	—	—
—	103	—	—	—
—	112½	75·5	<0·05 >0·04	<0·55 >0·41
			a.m.	**0·47**
3·65	0 = 112½ + ε	51·0 (?)	—	—
—	112⅝	—	26·0	3·25
—	112¾	44·0	—	—
—	112⅞	—	8·0	3·0
—	113	42·0	—	—
—	113½	—	2·5	2·5
—	114	39·5	—	—
—	114½	—	1·5	3·0
—	115	38·0	—	—
—	115½	—	1·0	3·0
—	116	37·0	—	—
—	118½	—	0·5	3·0
—	121	34·5	—	—
—	123½	—	0·3	3·3
—	126	33·0	—	—
—	132	—	0·17	3·3
—	138	31·0	—	—
—	143	—	0·1	3·0
—	148	30·0	—	—
—	153	—	—	—
—	158	29·0	>0·075 <0·1	<4·0 >3·0
			a.m.	**3·12**

TABLE XI.—*Experiment* 9 (December 2, 1904).

P	τ	L	$\frac{\Delta L}{\Delta \tau}$	$\frac{\Delta L}{\Delta \tau} \times \tau$
3·65	0·0 × 60	0·0	—	—
33·65	0·0 + ϵ	95·0 (?)	—	—
—	$\frac{1}{32}$	—	128·0	4·0
—	$\frac{1}{16}$	103·5	—	—
—	$\frac{3}{32}$	—	48·0	4·5
—	$\frac{1}{8}$	106·5	—	—
—	$\frac{3}{16}$	—	24·0	4·5
—	$\frac{1}{4}$	109·5	—	—
—	$\frac{3}{8}$	111·0	8	
—	$\frac{1}{2}$	112·0	— } 8·0	4·0
—	$\frac{5}{8}$	113·0	8	
—	$\frac{3}{4}$	113·5	4	
—	1	114·5	— } 4·0	4·0
—	$1\frac{1}{4}$	115·5	4	
—	$1\frac{1}{2}$	116·5	2	
—	2	117·5	— } 2·0	4·0
—	$2\frac{1}{2}$	118·5	2	
—	$3\frac{1}{2}$	119·5	—	—
—	4	—	1·0	4·0
—	$4\frac{1}{2}$	120·5	—	—
—	$6\frac{1}{2}$	—	0·75	4·8
—	$8\frac{1}{2}$	123·5	—	—
—	11	—	0·4	4·4
—	$13\frac{1}{2}$	125·5	—	—
—	15	—	0·5 (?)	—
—	$16\frac{1}{2}$	$127{\cdot}0^{+}$	—	—
—	$18\frac{1}{2}$	—	0·25	4·6
—	$20\frac{1}{2}$	$128{\cdot}0^{+}$	—	—
—	$20\frac{5}{8}$	$128{\cdot}0^{+}$	—	—
	ʌʌʌʌʌ		a.m.	**4·28**
33·65	0 = $21\frac{1}{4}$	127·0	—	—
—	$21\frac{5}{8}$	—	3·3	1·25
—	22	129·5	—	—
—	$22\frac{1}{2}$	—	1·0	1·25
—	23	130·5	—	—
—	24	—	0·5	1·37
—	25	$131{\cdot}5^{-}$	—	—
—	$27\frac{1}{2}$	—	0·2	1·25
—	30	132·5	—	—
—	$35\frac{1}{2}$	—	0·09	1·26
—	41	133·5	—	—
	ʌʌʌʌʌ		a.m.	**1·28**
33·65	0 = $41\frac{3}{4}$	132·0	—	—
—	$42\frac{7}{8}$	—	0·9	1·0
—	44	134·0	—	—
—	$45\frac{3}{4}$	—	0·3	1·14
—	$47\frac{1}{2}$	135·0	—	—
—	53	—	—	—
—	$58\frac{1}{2}$	136·0	0·09	1·0
—	$61\frac{1}{5}$	$136{\cdot}0^{+}$	—	—
			a.m.	**1·04**

TABLE XI.—*continued.*

P	τ	L	$\frac{\Delta L}{\Delta \tau}$	$\frac{\Delta L}{\Delta \tau} \times \tau$
	ΛΛΛΛΛ			
38 65	0 = 61¾	134·5		—
—	62¼	—	1·0	0 88
—	63½	136·0^{+}	—	—
—	65½	—	0·25	0·94
—	67½	137·0	—	—
—	74½	—	0·7	0·89
	81½	138·0	—	—
—	81¾	108 0^{+}	—	—
	ΛΛΛΛΛ			a.m. **0·90**
38 65	0 = 83	137·5^{+}	—	—
—	85	—	0·25	0 5
—	87	138·5	—	—
—	91	—	0 06	0 5
—	95	139 0	—	—
—	102	139·0^{+}	—	—
	ΛΛΛΛΛ			a.m. **0·5**
38 65	0 = 103	138·25	—	—
—	104	—	0 75	0·37
—	105	139·0	—	—
—	111	—	0·42	0 39
—	117	139 5	—	—
—	122½	139 5^{+}	—	—
				a.m. **0·36**
3 65	0 = $122\frac{9}{10}$	84·5 (?)	—	—
—	123	76·0	—	—
—	123¼	—	12·0	4·2
—	123½	70·0	—	—
—	124	—	4·0	4·4
—	124½	66 0	—	—
—	125	—	2·0	4·2
—	125½	64·0	—	—
—	126½	—	1·25	4·5
—	127½	61·5	—	—
—	130	—	0·6	4·3
—	132½	58·5	—	—
—	137½	—	0 3	4 35
—	142½	55·5	—	—
—	152½	—	0·15	4·5
—	162½	52·5	—	—
—	170	—	$<0{\cdot}1$	$<4{\cdot}7$
—	177½	51·0^{+}	$>0{\cdot}08$	$>3{\cdot}9$
				a.m. **4·34**

TABLE XII.—*Experiment* 10 (December 5, 1904)

P	T	L	$\frac{\Delta L}{\Delta T}$	$\left[\frac{\Delta L}{\Delta T}\right]$ Extrapolation	$\frac{\Delta L}{\Delta T} \times T$
3·1	0·0 × 60	0·0	—	—	—
23·1	0·0 + ϵ	75·0	—	—	—
—	⅛	—	28·0	—	3·5
—	¼	82·0	—	—	—
—	⅜	—	8·0	—	3·0
—	½	84·0	—	—	—
—	¾	—	4·0	—	3·0
—	1	86·0^{+}	—	—	—
—	1½	—	2·5	—	3·75
—	2	88·5	—	—	—
—	2½	—	1·5	—	3·75
—	3	90·0^{-}	—	—	—
—	4	—	0·75	—	3·0
—	5	91·5	—	—	—
—	6	—	0·5	—	3·0
—	7	92·5	—	—	—
—	8½	—	0·35	—	3·0
—	9⁹⁄₁₀	93·5			
	^^^^^		2·3		**a.m. 3·25**
	11	96·0			
—	13½	—	0·2	0·24	3·25
—	16	97·0	—	—	—
—	18	—	0·25	0·18	3·25
—	20	98·0	—	—	—
—	20¼	98·0^{+}			
	^^^^^		0·6	—	—
—	21½	99·0			
—	23¼	—	0·11	0·14	3·25
—	26	99·5	—	—	—
—	28½	—	0·2	0·11	3·25
—	31¹⁄₁₀	100·5			
	^^^^^		0·36	—	—
—	32½	101·0			
—	37	—	0·0	0·09	3·25
—	41½	101·0			
	^^^^^		0·17	—	-
—	43	101·25			
—	47	—	0·0	0·07	3·25
—	51½	101·25			
	^^^^^		0·33	—	—
—	53	101·75			
—	57½	—	0·11	0·06	3·25
—	60	102·5	—	—	—

TABLE XIII.—*Experiment* 11 (December 29, 1904).

P	τ	L	$\frac{\Delta L}{\Delta \tau}$	$\frac{\Delta L}{\Delta \tau} \times \tau$
4·8	0·0 × 60	0·0	—	—
7·8	1	—	—	—
14·8	2	—	—	—
24·8	3	—	—	—
44·8	4	—	—	—
—	$5\frac{1}{5} - \epsilon$	105·0	—	—
74·8	$0 = 5\frac{1}{5}$	130·5 (?)	—	—
—	$5\frac{9}{10}$	—	22·5	2·25
—	6	135·0	—	—
—	$6\frac{1}{8}$	—	7·0	2·27
—	$6\frac{1}{4}$	136·75	—	—
—	$6\frac{1}{6}$	—	4·0	2·3
—	$6\frac{1}{2}$	137·75	—	—
—	$6\frac{5}{8}$	—	3·0	2·47
—	$6\frac{3}{4}$	138·5	—	—
—	$7\frac{1}{8}$	—	2·33	3·1
—	$7\frac{1}{2}$	140·25	—	—
—	$7\frac{3}{4}$	—	2·5	3·62
—	8	141·5	—	—
—	$8\frac{1}{2}$	—	—	—
—	9	142·75+	<1·5 >1·25	<4·0 >3·37
—	10	—	0·87	3·67
—	11	144·5	—	—
—	12	—	0·62	3·87
—	13	145·75	—	—
—	14	—	0·5	4·1
—	15	146·75	—	—
—	17	—	0·44	4·8
—	19	148·5	—	—
—	21	—	0·37	5·62
—	23	150·0	—	—
—	25	—	0·31	5·94
—	27	151·25	—	—
—	31	—	0·25	6·25
—	35	153·25	—	—
—	40	—	0·20	6·8
—	45	155·25	—	—
—	50	—	0·15	6·6
—	55	156·75	—	—
—	60	—	0·12	6·77
—	65	158·0	—	—
—	65·5	158·0	—	—

TABLE XIV.—*Experiment* 12 (October 31, 1904).

τ	P	$\frac{\Delta P}{\Delta \tau}$	L	$\frac{\Delta L}{\Delta P}$	$\frac{\Delta L}{\Delta \tau}$
0·0 × 60	14·15		0·0		
1	15 02	0·014	1 0	1·08	0 0
2	15 90	0 015	2 75	—	0·0155
3	16·77	0·014	5·0	—	0·0230
4	17 63	0·014	7 5	—	0·0273
6	19·33	0 014	11·75	—	0 0212
8	20 96	0·014	15·5	—	0 0177
10	22·51	0·013	19·0	—	0·0162
15	26·09	0 012	26·75	—	0 0139
20	29 31	0 011	33·5	—	0·0118
25	32·20	0·010	39 5	—	0 0101
30	34 85	0·009	40 75	—	0·0087
35	37·25	0·008	49·75	—	0·0087
—	—	0·007		—	(?)
40	39·40		(?)		
45	41·36	0 006	62·75	—	(?)
50	43 23	0·006	66·25	—	0 0054
55	44 98	0·006	69 5	—	0 0050
60	46 61	0 005	72·5	—	0 0046
65	48 11	0·005	75 0	—	0·0033
70	49 47	0·005	77 25	—	0·0030
75	50 75	0·004	79 0	—	0 0016
80	52 10	0·005	80·75	—	0·0013
126·5	72·00	0 007	—	1 06	0 0
			101·75	—	—
		a.m. **0·0076**			
126 5	72·00		101·75		
127	70·96	0·034	101·5	0·24	0 0
128	68·87	0·035	101·0	0·24	0·0

TABLE XV.—*Experiment* 18 (October 26, 1904).

τ	P	$\frac{\Delta P}{\Delta \tau}$	L	$\frac{\Delta L}{\Delta P}$	$\frac{\Delta L}{\Delta \tau}$
0 0 × 60	14·0	0·035	0·0	1·25	0·0
½	15·05	0·035	1·25 +	—	0·004
1	16 1	0 035	2·75	—	0 007
1½	17·14	0·034	4·25	—	0·015
2	18·17	0 034	6·0	—	0·024
2½	19·19	0 034	8·0	—	0·053
3	20·21	0·034	11·0	—	0·035
3½	21·22	0·033	13·25 +	—	0·029
4	22·22	0·033	15·50 −	—	0·022
5	24·20	0·032	19·25	—	0·021
6	26·15	0 030	23 0	—	0 019
8	29·93	0·029	30·0	—	0·016
10	33·56	0·028	36·5	—	0·014
12	37·08	0·027	42·5	—	0·011
14	40·43	0·027	48·0	—	0·009
16	43 69	0 026	53·25 −	—	0·007
18	46 85	0 026	58·0	—	0·005
20	49·93	0·025	62·5	—	0·004
22	52 93	0 024	66·75	—	0 003
24	55·85	0 024	70·75	—	0 0013
26	58·72	0 024	74·50	—	0·0012
28	61·55	0·023	78 25 −	—	0·0005
30	64 35	0 024	81·75	1·25	0·0
44½	85·82		108·0		
		a.m. 0·027			
44½	85·82	0·036	108·0	0·18	0·0
46	82·68	0·038	107·75		
47	80·42	0·038	107 25		
48	78·16	0·038	106·75		
49	75 90	0·038	106·25		
50	73·64	0·038	105·5		0·0057
52	69·12	0·038	104 25	—	0·0037
54	64·60	0·038	102 5	—	0 0078
56	60·09	0·037	100 75	—	0·0078
58	55·62	0 036	98·75	—	0·010
60	51·23	0 035	96·5	—	0·012
62	46 98	0·032	94·0	—	0·014
64	43·18	0·030	91·25	—	0·017
66	39·48	0 029	88·25	—	0·020
68	36·02	0 028	84·75	—	0·024
70	32·72	0·026	81·25	—	0·024
72	29 5	0 025	77·25	—	0·029
74	26 59	0·023	73 0	—	0·031
76	23·79	0·022	68·25	—	0·035
78	21·18	0·021	63·0	—	0·040
80	18 71	0·019	57·25	—	0·044
82	16·5	0·018	50·75	—	0 051
84	14·82	0·007	44 0	—	0·056
85	13·87	0 000	42·25	—	0·028
86	13·87		41·25	—	0·017
		a.m. 0·029			0·0063
88	13·87	—	40·25	—	0·0042
90	13·87	—	40·0	—	0·0038
95	13·87	—	39·0	—	0·0030
100	13·87	—	38·0 +	—	0·0027
105	13·87	—	37·25	—	0 0008
110	13 87	—	37·0	—	0 000
120	13·87		37 0		

TABLE XVI.—*Experiment* 14 (December 20, 1904). 1st *Tracing.*

τ	P	L	$\frac{\Delta}{\Delta P}$	$\frac{\Delta L}{\Delta \tau}$
0·0	10·75 + 0 0	0·0 −	0·44	0·0
0·14	0·42	0·25 −	—	2·3
0·28	0·85	0·75		
0·43	1·29	1·25	—	3·0
0·57	1·73	2·0	—	4·0'
0·71	2·16	2·75	—	4·0
0·85	2·59	3·75	—	5·8
1·0	3·04	5·0 −	—	7·6
1·14	3·47	6·5	—	8·8
1·28	3·89	8·25	—	11·8
1·42	4·32	10·5	—	14·8
1·71	5·20	14·75	—	13·4
2·0	6·08	18·5 −	—	11·6
2·28	6·93	22·0	—	10 9
2·56	7·78	25·25 −	—	10·5
2·71	8·24	27·0 −	—	9·8
3·66	11·13	37·0	—	9·25
4·61	14·02	+ 46·0	—	8·5
5·56	16·91	54·5	—	7·6
6·51	19·80	62·0	—	6·6
7·46	22·69	68·75	—	5·8
8·41	25·57	74·75	—	5·0
9·36	28·47	80·0	—	4·25
11·26	34·24	89·5	—	3 7
13·16	40·02	98·5	—	3·45
15·06	45·80	106·75	—	3·01
16·96	51·57	114·25	—	2·67
18·86	57·35	121·0	—	2·27
20·76	63·13	127·0	—	1·88
22·66	68·91	132·25	—	1·5
23·61	71·80	134·5	—	1·08
24·08	73·23	135·5	0·42	0·85
29·78	10·75 + 73·23	+ 139·75	—	—
				$\frac{\Delta L}{\Delta \tau} \times \tau$
0=31·35	10 75	38·25 (?)	—	—
31·82	—	—	—	1·91
32·3	—	34·5	—	—
33·1	—	—	—	1·84
34·2	—	32·5	—	—
36·1	—	—	—	1·95
38·0	—	31·0	—	—
40·35	—	—	—	1·92
42·7	—	30·0	—	—
45·1	—	—	—	2·14
47·5	—	29·25	—	—
49·87	—	—	—	1·95
52·25	—	28·75	—	—
54·62	—	—	—	2·11
57·0	—	+ 28·25	—	—
59·37	—	—	—	1·83
61·75	—	28·0	—	—
600·0 by extrapolation	—	22·5	—	—
			a.m.	**1·98**

TABLE XVII.—*Experiment* 14 (December 20, 1904). *6th Tracing.*

τ	P	L	$\frac{\Delta L}{\Delta P}$	$\frac{\Delta L}{\Delta \tau}$
0·0	10·75 + 0·0	0·0		
0·14	0·44	0·25^{+}	0·68	0·0
0·28	0·89	1·0^{+}	—	3·4
0·48	1·35	2·0^{+}	—	4·9
0·57	1·81	3·25^{-}	—	6·0
0·71	2·27	4·5^{-}	—	6·9
0·86	2·73	5·75	—	7·3
1·0	3·18	7·25^{-}	—	8·3
1·14	3·62	8·75	—	9·4
1·42	4·52	12·25	—	10·8
1·71	5·44	16·25	—	12·1
2·0	6·36	19·5	—	9·3
2·28	7·26	22·25^{-}	—	7·7
2·57	8·17	24·75^{-}	—	7·0
2·71	8·62	25·75^{+}	—	6·1
3·67	11·67	33·0	—	5·5
4·63	14·72	39·75	—	5·1
5·59	17·78	46·0	—	4·6
6·55	20·83	51·75	—	4·1
7·51	23·88	56·75	—	3·3
8·47	26·93	61·5	—	3·0
9·43	29·99	66·0	—	2·75
11·35	36·09	74·25	—	2·4
13·27	42·2	82·0	—	2·11
15·9	48·3	88·5	—	1·46
17·1	54·36	93·75	—	0·81
19·0	60·42	98·75	—	0·68
20·9	66·5	103·0	—	0·30
22·8	72·5	107·25^{-}		
24·7	78·58	111·25	0·61	—
25·17	80·04	112·25		
30·4	10·75 + 80·04	113·5^{+}	—	0·25
				$\frac{\Delta L}{\Delta \tau} \times \tau$
0=33·0	10·75	11·5 (?)	—	—
33·6	—	—	—	1·5
34·2	—	8·5	—	—
36·1	—	—	—	1·63
38·0	—	6·5	—	—
42·75	—	—	—	1·54
47·5	—	5·0	—	—
52·25	—	—	—	1·52
57·0	—	4·25	—	—
600·0 by extrapolation	—	0·0	—	—
				a.m. 1·56

TABLE XVIII —*Experiment* 14 (December 20, 1904). 10*th Tracing.*

τ	P	L	$\frac{\Delta L}{\Delta P}$	$\frac{\Delta L}{\Delta \tau}$
0 0	10 75 + 0·0	0·0		
0·14	0·44	0·25^{+}	0·71	0 0
0 28	0·89	1·0	—	2·6
0·43	1·34	1·75	—	2 7
0·57	1·77	2·5	—	3 0
0·71	2·21	3·25	—	3 0
0·85	2 64	4·0^{+}	—	3·5
1·0	3 09	5 0	—	4 0
1·14	3·54	6·0	—	4 7
1·28	3·98	7·0^{+}	—	5·3
1 42	4 43	8 25	—	6·0
1·57	4·88	9·5^{+}	—	6·5
1 71	5·32	11·0^{+}	—	8·4
2·0	6·20	14·75	—	10 4
2·28	7 09	19 0	—	12·8
2 56	7·98	22·0	—	8 5
2·85	8·86	25·0^{-}	—	7 7
3·8	11·82	32·25	—	5·3
4 75	14·77	39·0	—	4 8
5·7	17·73	45 25	—	4 25
6 65	20·68	51·0	—	3 7
7·6	23·64	56 0	—	3 45
8 55	26·59	61·75	—	3·2
9·5	29·54	66 25	—	2 4
10·45	32 56	70·5	—	2 15
11·4	35·45	74·0	—	1 35
13 3	41·36	80·0	—	0 83
15·2	47·27	85 5	—	0 56
17·1	53·18	90·75	—	0·43
19·0	59·10	95 75	—	0·30
19·95	62·05	98 0	0 75	0·0
20 9	65 0	100·25		0 0
22 8	70·91	104·75		0 0
24·7	76·82	109 25		0 0
25·17	78·28	110·25^{+}		0 0
				$\frac{\Delta L}{\Delta \tau} \times \tau$
31·35	10·75+78 28	110 25^{+}	—	—
0=34·5	10·75	8 25	—	—
41·0	—	—	—	1·12
47·0	—	5·75	—	—
52·0	—	—	—	1 28
57 0	—	5 0	—	—
600·0 by extrapolation	—	1 25	—	—
			a.m.	**1·20**

TABLE XIX.—*Experiment* 14 (December 20, 1904).

	τ	$\frac{\Delta P}{\Delta \tau}$	$\frac{\Delta L}{\Delta P}$	$\frac{\Delta L}{\Delta \tau} \times \tau$
Exp. 1	24·08	3·04	0·42	1·95
2	23·38	3·17	0·48	1·80
3	25·51	3·17	0·51	1·70
4	24·87	3·10	0·56	1·67
6	25·17	3·18	0·61	1·56
7	24·90	3·12	0·70	1·42
9	25·36	3·11	0·75	1·12
10	25·17	3·11	0·75	1·20
11	26·22	3·06	0·77	1·13

TABLE XX.—*Experiment* 15 (December 15, 1904).

Exp. 1	59·85	1·17	0·32	1·89
2	62·70	1·18	0·37	1·82
5	63·65	1·17	0·53	1·57
7	65·55	1·14	0·62	1·47
9	63·65	1·20	0·64	1·30
10	66·50	1·17	0·65	1·19

TABLE XXI.—*Experiment* 16 (December 14, 1904).

Exp. 1	97·85	0·65	0·34	1·88
2	102·60	0·66	0·44	1·79
3	104·50	0·66	0·51	1·66
5	114·00	0·63	0·60	1·52
6	114·95	0·64	0·64	1·41
8	113·00	0·67	0·70	1·20

(*B*) *The Temperature Variable.*

TABLE XXII.—*Experiment* 1 (December 18, 1906).

τ	T	$\frac{\Delta T}{\Delta \tau}$	L
0	12·0	—	0·00
2	12·0	—	0·00
4	12·0		+ 0·05
6	—		+ 0·05
8	—	0·50	+ 0·05
10	—		0·00
12	16·0		0·00
14	—	0·28	0·00
16	17·1	0·35	− 0·05
18	17·8		0·00
20	18·0	0·10	+ 0·05
22	—	0·15	+ 0·05
24	18·6	0·20	+ 0·05
28	19·4	0·22	+ 0·05
32	20·3	0·25	+ 0·05
36	21·3	0·32	+ 0·05
40	22·6	0·35	+ 0·05
44	24·0	0·35	+ 0·05
48	25·4	0·40	+ 0·05
52	27·0	0·45	0·00
56	28·8	0·40	− 0·05
60	30·4		− 0·10
64	32·0	0·33	− 0·20
68	33·3	0·35	− 0·40
72	34·7	0·25	− 0·80
74	35·2	0·35	− 1·10
76	35·9	0·20	− 1·40
78	36·3	0·35	− 1·85
80	37·0	0·25	− 2·55
82	37·5	0·25	− 3·35
84	38·0	0·25	− 4·70
86	38·5	0·25	− 7·10
88	39·0	0·25	− 10·35
90	39·5		− 15·85
92	—	0·12	− 22·65
94	40·00	—	− 27·60
96	—	0·23	− 32·30
98	40·9	—	− 35·60
100	—	0·25	− 38·10
102	41·9	0·23	− 39·90
106	42·8	0·25	− 42·10
110	43·8	0·23	− 43·10
114	44·7		− 43·60

TABLE XXIII.—*Experiment* 2 (December 22, 1905).

τ	T	$\frac{\Delta T}{\Delta \tau}$	L
0	—	—	0·00
2	12·0	—	+ 0·10
4	—	0·10	+ 0·15
6	12·4	0 30	+ 0·20
8	13·0	0·35	+ 0·25
10	13·7	0·20	+ 0·25
12	14·1	0·60	+ 0·25
14	15·3	0 55	+ 0·25
16	16·4	0·50	+ 0·25
18	17·4	0·50	+ 0·20
20	18·4	0·45	+ 0·20
22	19·3	0 50	+ 0·20
24	20·3	0·55	+ 0·15
26	21·4	0·60	+ 0·15
28	22·6	0·55	+ 0·20
30	23 7	0·55	+ 0·20
32	24·8	0·60	+ 0·20
34	26·0	0·45	+ 0·20
36	26 9	0·50	+ 0·20
38	27·9	1·00	+ 0·20
40	29·9	0·50	+ 0·15
42	30·9		0·00
43	—	0·60	− 0 10
44	31·9	—	− 0 15
45	—	0 55	− 0·20
46	33·0	—	− 0·20
47	—	0·40	− 0·25
48	33·8	—	− 0·30
49	—	0·60	− 0·60
50	35·0	0·50	− 0·70
51	35·5	0·50	− 1·00
52	36·0	1·10	− 1·40
53	37·1	0·30	− 2·15
54	37·4	0·30	− 2·95
55	37 7	0·40	− 3·70
56	38·1	0·60	− 5·00
57	38·7	0·70	− 6·70
58	39·4	0·50	− 9·75
59	40·1	0·30	− 12·30
60	40·4	0·20	− 14·70
61	40·6	0·30	− 16·30
62	40·9	0·40	− 19·20
63	41·3	0·50	− 22·50
64	41·8	1·00	− 27·20
65	42·8	1·00	− 34·70
66	43·8	0·90	− 42·20
67	44·7	0·30	− 46·90
68	45·0	0·70	− 48·20
69	45·7	0·80	− 48·70
70	46·5		− 48·50

TABLE XXIV.—*Experiment* 3 (December 13, 1906).

τ	T	$\frac{\Delta T}{\Delta \tau}$	L
0	10·8	0·05	0·00
2	10·9	0 20	0·00
4	11·3	0·35	0·00
6	12·0	0 45	0·00
8	12·9	0·15	+ 0·05
10	13·2	0·35	+ 0·05
12	13 9	0·35	+ 0·10
14	14·6		+ 0·15
16	—	0·42	+ 0·15
18	16·3	—	+ 0·15
20	—	0·55	+ 0·20
22	18 5	0·25	+ 0·25
24	19·0	0·45	+ 0·25
26	19·9	0·45	+ 0·30
28	20·8	0·30	+ 0·35
30	21·4	0·35	+ 0·35
32	22 1	0·45	+ 0·35
34	23·0	0·35	+ 0·35
36	23·7	0 35	+ 0·35
38	24·4	0·35	+ 0 35
40	25·1	0·40	+ 0·35
42	25·9	0·30	+ 0·35
44	26·5	0·40	+ 0·35
46	27·3	0·40	+ 0·35
48	28·1	0 35	+ 0·35
50	28·8	0·25	+ 0·35
52	29·3	0·15	+ 0·35
54	29·6	0·25	+ 0·35
56	30·1		+ 0·30
57	—	0·45	+ 0·30
58	31·0	—	+ 0·25
59	—	—	+ 0·10
60	31·7	—	+ 0·10
61	—	0·50	+ 0·05
62	32·7	—	+ 0 05
63	—	0·60	0·00
64	34·9	—	− 0 10
65	—	0·60	− 0·20
66	36·1	—	− 0·65
67	—	0·45	− 1·30
68	37·0	—	− 2·20
69	—	0·30	− 3·65
70	37 6	—	− 4·70
71	—	0·60	− 6·15
72	38·8	—	7·65
73	—	0·50	9 30
74	39 8	—	11·15
75	—	0·80	14·25
76	41·4	—	17·65
77	—	0·80	21·75
78	43·0	—	27·40
79	—	0·45	33·50
80	43·9	—	39·85
81	—	0 50	44·85
82	44·9	—	48 75
83	—	0·20	49·95
84	45·3	0·30	50·45
86	45·6		51·60

TABLE XXV.—*Experiment* 4 (December 13, 1906).

τ	T	$\frac{\Delta T}{\Delta \tau}$	L
0	15·0		0·00
1	15·6	0·6	+ 0·05
2	17·0	0·4	+ 0·05
3	18·3	0·7	+ 0·10
4	19·3	1·0	+ 0 05
5	20·4	1 1	+ 0·10
6	22·3	1·9	+ 0 05
7	24·6	1·8	—
8	26·8	2·2	+ 0·05
9	28·5	1·7	0·00
10	30·0	1·5	− 0·05
11	—	0·5	− 0 05
12	31·0		− 0·05
13	31·6	0·4	− 0·05
14	32·2	0 6	− 0 10
15	33·2	1·0	0 10
16	34·5	1·3	0·25
17	36·0	1·5	0·35
18	37·2	1·2	2·50
19	38·5	1·3	8·50
20	40·0	1·5	16·75
21	41·3	1·3	28·25
22	42 3	1 0	39·25
23	43 0	0 7	46·75
24	43 6	0·6	51 25
25	44·3	0·7	52·65
26	45·0	0·7	52·80
27	45·7	0·7	52·65

TABLE XXVI.—*Experiment* 5 (December 24, 1906).

τ	T	$\frac{\Delta T}{\Delta \tau}$	L
0	12·6		0 00
2	13·5	0·45	0·00
4	14·0	0·25	0·00
6	14·5	0·25	0·00
8	15·9	0·70	0 00
10	16·9	0 50	0.00
12	17·5	0·30	0·00
14	18·0	0·25	+ 0 05
16	18·5	0 25	+ 0 05
18	19 0	0·25	+ 0·05
20	20·0	0·50	+ 0·05
22	21·5	0·75	+ 0·05
24	23·0	0 75	+ 0·10
26	24·1	0·55	+ 0·10
28	25·8	0·85	+ 0 05
30	27·3	0·75	− 0·05
32	28·6	0·65	− 0 10
34	29·8	0·60	− 0 10
36	30·5	0 35	− 0·10
38	31·4	0 45	− 0 05
40	32·4	0·50	− 0·05
42	33·4	0 50	− 0 05
43	34·0	0·60	− 0·05
44	34·7	0·70	− 0 10
45	35·4	0 70	− 0·20
46	36·0	0·60	− 0 30
47	—	0·65	− 0·35
48	37 3	—	− 0 55
49	—	0·70	− 1·00
50	38 7	—	− 1·75
51	—	0 65	− 2·25
52	40·0	—	− 3·40
53	—	0 65	− 4·70
54	41 3	—	− 6·80
55	—	0·55	− 9 10
56	42·4	—	− 13·10
57	—	0·45	− 16 75
58	43·3	—	− 20·50
59	—	0·50	− 24·00
60	44 3	—	− 28 00
61	—	0·65	− 32·00
62	45·6	—	− 35·50
63	—	0·65	− 39 00
64	46·9	—	− 41·50
65	—	0·65	− 43·70
66	48·2	—	− 45·00
67	—	0·65	− 46·20
68	49 5		− 47·20
69	50·2	0·70	− 47 50
70	50 8	0·60	− 47·50

TABLE XXVII.—*Experiment* 6 (December 24, 1905).

τ	T	$\frac{\Delta T}{\Delta \tau}$	L
0·0	15·9		0·00
2·0	21·2	2·6	+ 0·10
4·0	24·7	1·7	+ 0·10
6·0	26·3	0·8	+ 0·15
7·5	26·3	0·0	+ 0·20
8·5	26·3	0·0	+ 0·20
9·5	27·1	0·8	+ 0·25
10·5	28·1	1·0	+ 0·30
11·5	29·9	1·8	+ 0·30
12·5	32·3	2·4	+ 0·30
13·5	34·3	2·0	+ 0·30
14·5	36·2	1·9	+ 0·05
15·0	37·1	0·9	− 0·25
15·5	38·6	3·0	− 1·75
16·0	39·6	2·0	5·25
16·5	40·4	1·6	11·25
17·0	41·6	2·4	19·75
17·5	42·7	2·2	25·75
18·0	43·8	2·2	31·75
18·5	45·2	2·8	38·75
19·0	46·0	1·6	42·65
19·5	46·7	1·4	45·45
20·0	47·3	1·2	46·75
20·5	47·8	1·0	47·55
21·5	48·3	0·5	47·65
23·0	50·0	0·7	47·55

TABLE XXVIII.—*Experiment* 7 (December 1, 1906).

τ	T	$\frac{\Delta T}{\Delta \tau}$	L
0	12·4		0·00
5	12·4	+ 0·0	0·00
10	12·4	+ 0·0	0·00
15	13·6	+ 0·24	0·00
17	14·4	+ 0·26	0·00
19	15·3	+ 0·45	0·00
20	15·8	+ 0·50	0·00
22	16·3	+ 0·25	0·00
24	17·0	+ 0·35	0·00
26	17·8	+ 0·40	0·00
28	18·5	+ 0·35	0·00
30	19·6	+ 0·55	0·00
32	20·7	+ 0·55	0·00
34	21·7	+ 0·50	0·00
36	22·7	+ 0·50	0·00
38	23·8	+ 0·55	− 0·05
40	24·7	+ 0·45	− 0·10
42	25·4	+ 0·35	− 0·15
44	26·4	+ 0·50	− 0·30
46	27·6	+ 0·60	− 0·35
48	29·2	+ 0·80	− 0·55
50	31·1	+ 0·85	− 0·95
52	32·5	+ 0·70	− 1·40
53	31·6	− 0·90	− 1·45
54	30·0	− 1·60	− 1·25
56	25·7	− 2·15	− 1·15
58	23·0	− 1·35	− 1·10
60	21·5	− 0·75	− 1·00
62	25·8	+ 2·25	− 1·05
64	28·8	+ 1·50	− 1·20
66	31·0	+ 1·10	− 1·70
68	33·5	+ 1·25	− 2·50
69	32·4	− 1·10	− 2·75
70	29·5	− 2·90	− 2·70
71	26·5	− 3·00	− 2·70
72	25·0	− 1·50	− 2·65
73	23·1	− 1·90	− 2·60
74	21·7	− 1·40	− 2·50
75	20·5	− 1·20	− 2·50
76	21·8	+ 1·30	− 2·50
77	24·5	+ 2·70	− 2·50
78	27·0	+ 2·50	− 2·50
79	29·0	+ 2·00	− 2·50
80	30·5	+ 1·50	− 2·60
81	31·5	+ 1·00	− 2·80
82	32·5	+ 1·00	− 3·00
83	33·2	+ 0·70	− 3·25
84	34·0	+ 0·80	− 3·55
85	34·4	+ 0·40	− 3·80
86	33·6	− 0·80	− 4·00
87	30·6	− 3·00	− 4·00
88	27·4	− 3·20	− 4·00
89	24·5	− 2·90	− 3·95
90	22·2	− 2·30	− 3·95
91	21·0	− 1·20	− 3·95
92	19·8	− 1·20	− 3·90
93	18·7	− 1·10	− 3·85
94	17·3	− 1·40	− 3·85
95	16·1	− 1·20	− 3·80
96	15·0	− 1·10	− 3·80

TABLE XXIX.—*Experiment* 8 (December 5, 1905).

τ	T	ΔT/Δτ	L	τ	T	ΔT/Δτ	L
0	13·0		+ 1·30	13	26·0		− 3·60
1	19·2	+ 6·2	+ 1·30	14	29·5	+ 3·5	− 3·65
2	23·0	+ 3·8	+ 1·30	15	32·5	+ 3·0	− 4·00
3	26·3	+ 3·3	+ 1·30	16	35·0	+ 2·5	− 4·35
4	29·0	+ 2·7	+ 1·30	17	32·0	− 3·0	− 5·60
5	32·0	+ 3·0	+ 1·30	18	28·5	− 3·5	− 5·60
6	34·0	+ 2·0	+ 0·30	19	24·5	− 4·0	− 5·50
7	36·3	+ 2·3	− 0·70	20	28·0	+ 3·5	− 5·50
8	38·0	+ 1·7	− 2·95	21	32·0	+ 4·0	− 5·60
9	32·0	− 6·0	− 3·70	22	35·0	+ 3·0	− 6·20
10	28·0	− 4·0	− 3·70	23	38·5	+ 3·5	− 8·70
11	25·0	− 3·0	− 3·65	24	41·5	+ 3·0	− 13·60
12	22·5	− 2·5	− 3·60				

TABLE XXX.—*Experiment* 9 (December 22, 1905).

τ	T	ΔT/Δτ	L	τ	T	ΔT/Δτ	L
0	24·4		+ 0·25	13·5	33·8	− 2·0	− 24·30
1	26·0	+ 1·6	+ 0·25	14	34·7	+ 1·8	− 25·25
2	28·5	+ 2·5	+ 0·25	15	37·7	+ 3·0	− 26·40
3	31·4	+ 2·9	0·00	16	41·3	+ 3·6	− 32·25
4	34·8	+ 3·4	− 0·60	17	44·4	+ 3·1	− 43·30
4·25	35·4	+ 2·4	− 0·95	17·5	45·0	+ 1·2	− 46·00
4·5	36·3	+ 3·6	− 1·35	18	40·0	− 10·0	− 49·30
4·75	37·1	+ 3·2	− 2·45	18·5	35·8	− 8·4	− 49·55
5	37·5	+ 1·6	− 3·00	19	34·9	− 1·8	− 49·50
6	33·2	− 4·3	− 2·40	19·5	36·5	+ 3·2	
7	31·4	− 1·8	− 2·30	20	37·5	+ 2·0	− 49·00
8	33·8	+ 2·4	− 2·30	21	40·2	+ 2·7	− 48·70
9	36·0	+ 2·2	− 2·30	21·5	42·0	+ 3·6	
10	37·9	+ 1·9	− 7·30	22	43·2	+ 2·4	− 48·50
11	39·5	+ 1·6	− 20·30	23	45·9	+ 2·7	− 49·40
12	40·2	+ 0·7	− 24·70	24	48·0	+ 2·1	− 50·20
12·5	38·0	− 2·2	− 24·30	24·5	49·5	+ 3·0	− 51·00
13	34·8	− 6·4	− 24·30	25	50·3	+ 1·6	− 51·00

[THE FOLLOWING TABLES SHOULD BE READ IN CONJUNCTION WITH CHAPTER II.]

TABLE XXXI.—*Experiment* 1 (September 15, 1899).

FIFTH EXTENSION CURVE.

τ	P	L	Tonus	$\left[C_2\left(a-\frac{\Delta L}{\Delta P}\right)\right]$	$[\text{Lgn.}\, C_1(P-\Delta)]$	Diff
0·0	0·0 + 1·86	$0{\cdot}0 \times 10^{-8}$	—	—	—	—
3·0	2·58	12·8	$7{\cdot}8 \times 10^{-8}$	− 0·850	− 0·850	0·000
6·2	5·22	30·8	6·5	− 0·708	− 0·701	+ 0·007
12·6	10·61	60·6	5·2	− 0·566	− 0·562	+ 0·004
25·4	21·89	102·1	3·9	− 0·425	− 0·426	− 0·001
33·5	33·50	121·3	—	—	—	—

$\Delta = 0{\cdot}3$ $C_1 = 4{\cdot}93 \times 10^{-3}$ $C_2 = -109.$

TABLE XXXII.—*Experiment* 2 (December 6, 1899).

τ	P	L	Tonus	$\left[C_2\left(a-\frac{\Delta L}{\Delta P}\right)\right]$	$[\text{Lgn.}\,C_1(P-\Delta)]$	Diff.
0·0	0·0 + 1·9	$0{\cdot}0 \times 10^{-3}$	—	—	—	—
3·0	2·33	13·1	$8{\cdot}4 \times 10^{-8}$	− 0·920	− 0·928	− 0·008
6·2	4·81	34·6	7·0	− 0·766	− 0·755	+ 0·011
12·6	9·77	67·9	5·6	− 0·613	− 0·605	+ 0·008
25·4	19·69	114·0	4·2	− 0·460	− 0·466	− 0·006
35·0	27·14	137·6	—	—	—	—

$\Delta = 0{\cdot}7$ $C_1 = 4{\cdot}45 \times 10^{-3}$ $C_2 = -109{\cdot}5$

TABLE XXXIII.—*Experiment* 3 (November 11, 1899).

τ	P	L	Tonus	$\left[C_2\left(a-\frac{\Delta L}{\Delta P}\right)\right]$	$[\text{Lgn.}\,C_1(P-\Delta)]$	Diff.
0·0	0·0 + 1·86	$0{\cdot}0 \times 10^{-8}$	—	—	—	—
3·0	2·56	17·0	$7{\cdot}2 \times 10^{-8}$	− 0·857	− 0·857	0·00
6·2	5·28	36·0	6·0	− 0·714	− 0·708	+ 0·006
12·6	10·74	64·9	4·8	− 0·571	− 0·599	− 0·028
25·4	21·64	104·0	3·6	− 0·428	− 0·434	− 0·006
30·9	26·33	116·0	—	—	—	—

$\Delta = 0{\cdot}3$ $C_1 = 4{\cdot}69 \times 10^{-8}$ $C_2 = -119$

TABLE XXXIV.—*Experiment* 4 (November 10, 1899).

τ	P	L	Tonus	Plastic.
0·0	1·88 + 0·0	0·00 × 10^{-3}	—	—
0·25	—	—	0·92 (?) × 10^{-3}	—
0·5	0·29	0·27	—	—
0·75	—	—	3·67	—
1·0	0·58	1·33	—	—
1·25	—	—	—	12·72 × 10^{-3}
1·5	—	—	4·59	—
1·75	—	—	—	12·94
2·0	1·16	3·99	—	—
2·5	—	—	8·25	13·09
3·0	1·74	7·98	—	—
4·6	—	—	—	11·90
6·2	3·60	20·22	9·00	—
9·4	—	—	—	10·68
12·6	7·81	43·62	7·20	—
19·0	—	—	—	8·53
25·4	14·74	75·55	5·40	—
33·4	—	—	—	7·07
41·4	24·01	103·74	3·60	—
42·4	24·57	105·20	—	—
	P = const			
42·5	1·88+24·57	105·34	—	—
43·25	—	—	—	0·77 × 10^{-3}
44·0	—	106·40	—	—
45·5	—	—	—	0·35
47·0	—	107·46	—	—
49·5	—	—	—	0·03
52·0	—	108·00	—	—
53·5	—	108·15	—	—
55·0	1·88+24·57	108·20	—	—
	Unloading of the muscle.			
55·0 + ϵ	1·88 + 0·0	13·78 (?)	—	—
56·5	—	12·77	—	—
57·0	—	—	—	− 0·53 × 10^{-3}
57·5	—	12·24	—	—
58·5	—	11·97	—	—
59·0	—	—	—	− 0·27
59·5	—	11·70	—	—

TABLE XXXV.—*Experiment* 4 (November 10, 1899).

τ	$\left[C_2\left(a - \frac{\Delta L}{\Delta P}\right)\right]$	[Lgn. $C_1 (P - \Delta)$]	Diff.
3·0	− 0·756	− 0·760	− 0·004
6·2	− 0·605	− 0·597	+ 0·008
12·6	− 0·454	− 0·452	+ 0·002
25·4	− 0·302	− 0·313	− 0·011
41·4	− 0·227	− 0·220	+ 0·007

$\Delta = 0·4 \quad C_1 = 13·2 \times 10^{-3} \quad C_2 = -84$

TABLE XXXVI.—*Experiment* 5 (December 2, 1899).

τ	P	L	Tonus	Plastic	$\frac{dl}{d\tau} \times \tau$
0·0	3·90 + 0·0	$0·00 \times 10^{-3}$	—	—	
0·25	—	—	$1·52(?) \times 10^{-3}$	—	
0·5	0·53	0·81	—	—	
0·75	—	—	3·04	—	
1·0	1·06	2·42	—	—	
1·25	—	—	—	$13·06 \times 10^{-3}$	
1·5	—	—	4·06	—	
1·75	—	—	—	15·62	
2·0	2·12	6·71	—	—	
2·5	—	—	4·94	16·00	
3·0	3·18	11·95	6·00	—	
4·6	—	—	—	14·44	
6·2	6·57	28·49	5·00	—	
9·4	—	—	—	12·76	
12·6	13·36	56·98	4·00	—	
18·8	—	—	—	9·88	
25·0	26·40	91·91	3·50	—	
25·2	27·00	91·87	—	—	
	P = const.				
25·2	3·90 + 27·00	91·87	—	—	
25·35	—	—	—	1·79	
25·5	—	91·91	—	—	
26·0	—	—	—	1·07	
26·5	—	92·99	—	—	
27·5	—	93·26	—	—	
28·5	—	—	—	0·18	
29·5	—	93·52	—	—	
45·2	Section of the nervus tibialis.				
50·0	—	101·05	—	—	
54·0	—	—	—	0·27	$2·88 \times 10^{-3}$
58·0	—	103·20	—	—	—
60·0	—	103·47	—	—	—
62·5	—	—	—	0·15	2·60
65·0	3·90 + 27·00	104·27	—	—	—
	Unloading of the muscle	$\left[\text{Elast. } 3·09 \times 10^{-3}\right]$			
65·0 + ϵ	3·90 + 0·0	20·94	—	—	
65·5	—	—	—	− 0·54	
66·0	—	20·41	—	—	
66·75	—	—	—	− 0·18	
67·5	3·90 + 0·0	20·14	—	—	

TABLE XXXVII.—*Experiment* 6 (December 3, 1899).

τ	P	L	Tonus	Plastic.	$\frac{dl}{d\tau} \times \tau$
0·0	1·90 + 0·0	0 0 × 10^{-8}	—	—	
0·375	—	—	3·36 × 10^{-8}	—	
0·75	0·72	2·42	—	—	
0·875	—	—	4·30	16·48 × 10^{-8}	
1	0·97	3·49	—	—	
1·25	—	—	5·60	15·88 (?)	
1·5	1·45	6·18	—	—	
1·75	—	—	6·72	16·90	
2·0	1·98	9·41	—	—	
2·25	—	—	7·13	17·36	
2 5	2·42	12·90	—	—	
2·75	—	—	6·72	15·36	
3·0	2·90	16·12	6·60	—	
4 6	—	—	—	14·51	
6·2	5·99	35·47	5·50	—	
9·4	—	—	—	12·60	
12·6	12 17	65·57	4·40	—	
19·0	—	—	—	9·74	
25·4	24·54	109·65	3·80	—	
26·27	—	—	—	7·90	
27·1	26·19	112·87	—	—	
	P = const.				
27·1	1·90 + 26·19	112·87	—	—	
27·5	—	113·14	—	—	
27·75	—	—	—	0·54	
28·0	—	113·41	—	—	
28·5	—	—	—	0·40	
29·0	—	113·81	—	—	
40·6	Section of the nervus tibialis.				
45·5	—	135·18			
46·0	—	—	—	1·07	5·91 × 10^{-8}
46·5	—	136·26	—	—	—
47·0	—	—	—	1·07	6·99
47·5	—	137·33	—	—	—
48·0	—	—	—	0·89	6·70
48·5	—	138·22	—	—	—
50·0	—	—	—	0·42	3·98
51·5	—	139·48	—	—	—
53·75	—	—	—	0·30	4·01
56·0	—	141·10			
56·3	Contraction.				
61·5	—	142·17	—	—	
63·5	—	144·59	—	—	
67·0	—	144·86	—	—	
70·0	—	144·86	—	—	
77·3	1·90 + 26·19	144·98	—	—	
	Unloading of the muscle	[Elast. 3 57 × 10^{-8}]			$\frac{dl}{d\tau} \times \tau$
77·3 + ϵ	1·90 + 0·0	51·46	—	—	
79·0	—	44·48	—	—	
79·5	—	—	—	− 1·48	− 2·35 × 10^{-8}
80·0	—	43·00	—	—	—
80·5	—	—	—	− 1·07	− 3·42
81·0	—	41·92	—	—	—
81·5	—	—	—	− 0·66	− 2·77
82 0	—	41·26	—	—	—
82·5	—	—	—	− 0·68	− 3·54
83·0	—	40·58	—	—	—
83·5	—	—	—	− 0·54	− 3·35
84·0	—	40·04	—	—	—
85·5	—	—	—	− 0·31	− 2·54
87·0	—	39·10	—	—	—
98·5	—	—	—	− 0·17	− 3·57
110·0	—	35·21	—	—	

a.m. 3·09

TABLE XXXVIII.—*Experiment* 6.

FIRST EXTENSION CURVE OF THE ATONIC MUSCLE.

τ	P	L	Plastic.	$\frac{dl}{d\tau} \times \tau$
132 = 0·0	1·9 + 0 0	0·00 × 10^{-3}	[Elast. 2·16 × 10·3^{-3}]	
0·5	0·5	1·07		
1·0	1·0	4·30	—	
1·25	—	—	4·29 × 10^{-3}	
1·5	1·5	7·52	—	
1·75	—	—	4·83	
2·0	2·0	11·02	—	
2·25	—	—	5·91	
2·5	2·5	15·05	—	
2·75	—	—	3·76	
3·0	3·0	18·01	—	
3·25	—	—	3·22	
3·5	3·5	20·69	—	
3·75	—	—	3·22	
4·0	4·0	23·38	—	
4·25	—	—	3 22	
4·5	4·5	26·07	—	
4·75	—	—	2·68	
5·0	5·0	28·49	—	
5 25	—	—	2·48	
5 5	5·5	30·91	—	
5·75	—	—	2·14	
6·0	6·0	33·06	—	
7·0	—	—	2·14	
8·0	8·0	41·66	—	
9·0	—	—	2·14	
10·0	10·0	50·26	—	
11·0	—	—	1 74	
12·0	12·0	58 05	—	
13·0	—	—	1·33	
14·0	14·0	65·04	—	
15·0	—	—	1·07	
16·0	16 0	71·49	—	
17·0	—	—	0 80	
18·0	18·0	77·40	—	
19·0	—	—	0·20 (?)	
20·0	20·0	82 77	—	
21·0	—	—	0·72	
22·0	22·0	87·88	—	
23·0	—	—	0·26	
24 0	24·0	92·72	—	
25·0	—	—	0·18 (?)	
26·0	26·0	97·29	—	
27·0	—	—	0·13 (?)	
28·0	28·0	101·86	—	
28·2	28·2	102·12	—	
	P = const.			
28·2	1·9 + 28·2	102·12	—	
30·1	—	—	0·28	
32·0	1 9 + 28·2	103·20	—	
	Unloading of the muscle.			
32 0 + ε	1·9 + 0·0	32·25 (?)	—	
34·0	—	11·29	—	
35·5	—	—	0·98	− 3·45 × 10^{-3}
37·0	—	8·33	—	—
39·0	—	—	0·47	− 3·29
41·0	—	6·45	—	—
43 0	—	—	0·34	− 3·70
45·0	—	5·11	—	—
48·0	—	—	0·18	− 2·88
51·0	—	4 03	—	—
56·0	—	—	0·13	− 3·22
61·0	—	2·69	—	—
66·0	—	—	0·08	− 2·75 ⎫ 3·29
71·0	—	1·88	—	— ⎪
77·5	—	—	0·08	− 3·82 ⎭
216 = 84·0	1·9 + 0·0	0·81	—	—

a.m. 3·30

TABLE XXXVIII.—*continued.*

SECOND EXTENSION CURVE OF THE ATONIC MUSCLE

τ	P	L	Plastic.	
216 = 0·0	1 9 + 0·0	0·00 × 10^{-3}	[Elast. 2 09 × 10^{-3}]	
0·5	0·45	0 94		
0·75	—	—	3·74	
1·0	0 90	3·76	—	
1 5	—	—	4·02	
2·0	1·80	9 67	—	
3·0	—	—	3 75	
4 0	3 60	20 96	—	
5·0	—	—	3 22	
6 0	5·40	31 17	—	
7 0	—	—	2 95	
8 0	7 20	40 85	—	
9 0	—	—	2 41	
10·0	9·00	49·45	—	
11·0	—	—	1 87	
12·0	10·80	56·97	—	
13·0	—	—	1 60	
14·0	12·60	63 96	—	
15 0	—	—	1 33	
16 0	14·40	70·41	—	
17·0	—	—	1·07	
18·0	16·20	76·32	—	
19 0	—	—	0·93	
20·0	18·00	81 97	—	
21·0	—	—	0·66	
22 0	19 8	87 07	—	
23·0	—	—	0 38	
24·0	21·60	91 64	—	
25·0	—	—	0 41	
26 0	23 40	96 21	—	
27·0	—	—	0 26	
28·0	25 20	100 51	—	
29 0	—	—	0 26	
30·0	27·00	104 81	—	
31 8	28·62	108·57	—	
	P = const			
31 8	1·9 + 28 62	108 57	—	
33·5	—	—	0 21	
35 2	1 9 + 28·62	109 25	—	
	Unloading of the muscle.			
35·2 + ϵ	1 9 + 0 0	36·14 (?)	—	$\frac{dl}{d\tau} \times \tau$
37 0	—	10·34	—	
38 0	—	—	0 87	− 2 44 × 10^{-3}
39·0	—	8·60	—	—
40·0	—	—	0 31	− 1·51
41 0	—	7 65	—	—
42 0	—	—	0 33	− 2·26
43·0	—	6·99	—	—
44 9	—	—	0 27	− 2 37
45·0	1·9 + 0·0	6·45	—	—

a.m. 2·16

TABLE XXXIX.—*Experiment* 7 (December 1, 1899).

τ	P	L	Tonus		Plastic.	$\frac{dl}{d\tau} \times \tau$
0·0	3·95 + 0·0	$0{\cdot}0 + 10^{-3}$	—		—	
0·25	—	—	$2{\cdot}69 \times 10^{-3}$		—	
0·5	0·60	1·61	—		—	
0·75	—	—	4·93		$32{\cdot}28 \times 10^{-3}$	
1·0	1·20	4·57	—		—	
1·5	—	—	7·17		32·00	
2·0	2·40	13·17	—		—	
2·25	—	—	7·94		—	
2·5	3·00	18·01	—		31·88	
2·75	—	—	8·96			
3·0	3·61	23·38	—	8·51		
3·25	—	—	8·06			
3·5	4·21	28·22	—		—	
5·2	6·25	43·27	—		—	
5·7	—	—	6·72			
6·2	7·45	51·38	—	6·49	—	
6·7	—	—	6·27		25·56	
7·2	8·65	58·86	—		—	
11·6	13·94	86·00	—		—	
12·1	—	—	4·48			
12·6	15·14	91·37	—	4·48	—	
13·1	—	—	4·48		21·18	
13·6	16·34	96·75	—		—	
20·0	24·04	125·24	—		—	
20·5	—	—	3·13			
21·0	25·24	129·00	—	3·02	—	
21·5	—	—	2·91		16·61	
22·0	26·44	132·49	—		—	
22·8	3·95 + 27·40	134·91	—		—	
	P = const.					
22·8	3·95 + 27·40	134·91	—		—	
23·9	—	—	—		0·98	
25·0	3·95 + 27·40	137·06	—		—	
	Unloading of the muscle.					
25·0 + ε	3·95 + 0·0	64·23 (?)	—		—	
36·0	—	56·71	—		—	
42·0	—	56·71	—		0·00	
46·0 − ε	—	56·71	—		—	
	Section of the nerve.					
46·0	—	−9·14	—		—	
46·125	—	—	—		10·75	$1{\cdot}34 \times 10^{-3}$
46·25	—	−6·45	—		—	—
46·875	—	—	—		4·30	1·61
46·50	—	−5·37	—		—	—
46·75	—	—	—		2·70	2·02
47·0	—	−4·03	—		—	—
47·5	—	—	—		1·61	2·42
48·0	—	−2·42	—		—	—
49·0	—	—	—		0·54	1·61
50·0	—	−1·34	—		—	—
53·7	—	—	—		0·18	1·40
57·4	—	0·0	—		—	—

TABLE XL.—*Experiment* 7.

FIRST EXTENSION CURVE OF THE ATONIC MUSCLE.

T	P	L	Plastic.
57·4 = 0	3·95 + 0·00	$0{\cdot}00 \times 10^{-3}$	[Elast. $2{\cdot}40 \times 10^{-3}$]
0·25	—	—	0·18
0·5	0·41	1·07	—
0·75	—	—	3·08
1·0	0·83	4·03	—
1·5	—	—	4 05
2·0	1·65	10·48	—
2·5	—	—	5 39
3 0	2·48	18·27	—
3·5	—	—	5·93
4·0	3·31	26 61	—
5 0	—	—	9·14
6·0	4 96	42·73	—
7·0	—	—	6·25
8·0	6·62	58·05	—
9·0	—	—	4·85
10·0	8·27	72·56	—
12·5	—	—	3·40
15·0	12·41	101·59	—
17 5	—	—	2·00
20·0	16·54	123·62	—
22·5	—	—	0 82
25·0	20·68	139·75	—
27·5	—	—	0·56
30·0	24 82	154·53	—
32·3	—	—	0·34
34·6	28·62	167·16	—
—	P = const.	—	—
34·6	3·95 + 28·62	167·16	—
35·8	—	—	0 45
37 0	3·95 + 28 62	168·24	—
		Unloading of the muscle	[Elast. $2{\cdot}80 \times 10^{-3}$]
$37{\cdot}0 + \epsilon$	3·95 + 0·00	88·15	—

SECOND EXTENSION CURVE OF THE ATONIC MUSCLE.

T	P	L	Plastic.
179·4 = 0	3·95 + 0·00	$0{\cdot}00 \times 10^{-3}$	[Elast. $2{\cdot}40 \times 10^{-3}$]
0·25	—	—	0·00
0·5	0·67	1·34	—
0·75	—	—	0·40
1·0	1·33	2·69	—
1 5	—	—	0 54
2·0	2·67	5 91	—
2·5	—	—	1·63
3·0	4·00	10 21	—
3·5	—	—	4·05
4·0	5·33	16·93	—
5·0	—	—	4 17
6·0	8·00	30·64	—
7·0	—	—	3·78
8 0	10·66	43·54	—
9 0	—	—	2 16
10·0	13·33	53·21	—
12·5	—	—	1·84
15·0	20·00	75·79	—
17·5	—	—	0·98
20·0	26·66	94·06	—
22·1	—	—	0·65
24·2	32 26	108·04	—
	P = const.		
24·2	3·95 + 32·26	108·04	—
25·8	—	—	0·67
27·4	3·95 + 32·26	110·19	—
		Unloading of the muscle	[Elast. $2\ 43 \times 10^{-3}$ (?)].
$27{\cdot}4 + \epsilon$	3·95 + 0·00	31·71 (?)	—

TABLE XLI.—*Experiment* 8 (December 4, 1900).

τ	P	L	Tonus	Plastic.
0·0	38 + 0·0	0·0 × 10^{-3}	—	—
0·5	—	—	0·45 × 10^{-3}	—
1·0	2·78	1·25	—	—
1·5	—	—	0·45	4·3 × 10^{-3}
2·0	5·57	2·5	—	—
2·5	—	—	0·54	4·3
3·0	8·35	4·0	—	—
3·5	—	—	0·63	4·5
4·0	11·14	5·75	—	—
4·6	—	—	—	7·9
5·2	14·48	8·0	—	—
6·2	17·27	10·0	0·76	2·7 (?)
7·2	20·05	12·25	—	—
9·4	—	—	—	4·6
11·6	32·31	22·0	—	—
12·6	35·09	24·25	0·80	4·5
13·6	37·88	26·5	—	—
25·0	69·62	50·5	—	—
26·0	72·41	52·5	0·72	0·7 (?)
27·0	75·19	54·5	—	—
33·0	—	—	—	3·9
39·0	108·61	77·25	—	—
40·0	111·40	79·25	0·67	4·0
41·0	114·18	81·0	—	—
43·5	—	—	—	—
45·8	38+127·55	89·0	—	—
	P = const.			
45·8	38+127·55	89·0	—	—
48·0	—	—	—	0·38
50·4	38+127·55	90·75	—	—
			Unloading of the muscle	[Elast. 0·47 × 10^{-3}]
50·4 + ϵ	38+ 0·0	30·5	—	—

Elasticity of the muscle during the interval (0—1″) = 0·45 × 10^{-3}

Elasticity of the muscle at the unloading = 0·47 × 10^{-3}

τ	$C_2\left(a - \frac{\Delta L}{\Delta P}\right)$	Lgn. C_1 (P − Δ)	Diff.
12·6	− 1·347	− 1·351	− 0·004
26·0	− 1·212	− 1·213	− 0·001
40·0	− 1·128	− 1·131	− 0·003

Δ 0·6 $C_1 = 0{\cdot}023 \times 10^{-3}$ $C_2 = -1684$

Length of the muscle, 11 cm.
Weight of the muscle, 37·5 grm.

TABLE XLII.—*Experiment* 8 (December 4, 1900).

τ	P	L	Tonus	Plastic.
0·0	38 + 0·0	0·0 $\times 10^{-3}$	—	—
0·5	—	—	0·34 $\times 10^{-3}$	—
1·0	2·94	1·0	—	—
1·5	—	—	0·43	4·5 $\times 10^{-3}$
2·0	5·88	2·25	—	—
2·5	—	—	0·51	4·4
3·0	8·82	3·75	—	—
3·5	—	—	0·68	4·9
4·0	11·76	5·75	—	—
4·6	—	—	—	5·0
5·2	15·28	8·5	—	—
6·2	18·22	10·75	0·81	6·6
7·2	21·16	13·25	—	—
11·6	34·09	23·25	—	—
12·6	37·03	25·5	0·72	4·6
13·6	39·97	27·5	—	—
19·5	—	—	—	4·4
25·0	73·47	51·75	—	—
26·0	76·41	53·75	0·64	2·4 (?)
27·0	79·35	55·5	—	—
33·0	—	—	—	4·1
39·0	114·62	77·0	—	—
40·0	117·56	78·75	0·60	4·4
41·0	120·50	80·5	—	—
45·5	—	—	0·60	—
49·9	38+146·61	95·75	—	—
	P = const.			
49·9	38+146·61	95·75	—	—
52·0	—	—	—	0·7
54·0	38+146·61	97·25	—	—
			Unloading of the muscle	[Elast. 0·89 $\times 10^{-3}$]
54·0 + ϵ	38 + 0·0	89·5	—	—

Elasticity of the muscle during the interval (0—1") = 0·34 $\times 10^{-3}$

Elasticity of the muscle at the unloading = 0·89 $\times 10^{-3}$

τ	$C_2\left(a - \frac{\Delta L}{\Delta P}\right)$	Lgn. C_1 (P − Δ)	Diff.
6·2	− 1·411	− 1·400	+ 0·011
12·6	− 1·254	− 1·261	− 0·007
26·0	− 1·115	1·123	− 0·013
40·0	− 1·045	− 1·042	+ 0·003

$\Delta = 0·6$ $C_1 = 0·084 \times 10^{-3}$ $C_2 = -1742$

TABLE XLIII.—*Experiment* 9 (December 6, 1900).

τ	P	L	Tonus	Plastic.
0·0	38 + 0·0	0 0 × 10^{-3}	—	—
0·5	—	—	0·20 × 10^{-3}	—
1·0	2·51	0·5	—	—
1·5	—	—	0·40	4·4 × 10^{-3}
2·0	5·02	1·5	—	—
2·5	—	—	0·50	4·5
3·0	7·53	2·75	—	—
3·5	—	—	0·50	4·3
4·0	10·04	4·0	—	—
4·6	—	—	—	4·5
5 2	13·06	5·75	—	—
6·2	15·57	7·25	0·60	9·4
7·2	18·08	8·75	—	—
11·6	29·13	16 25	—	—
12 6	31·64	18·0	0·70	4·3
13·6	34·15	19·75	—	—
25·0	62 78	38·75	—	—
26·0	65·29	40·25	0·60	3·3
27·0	67·80	41·75	—	—
53 0	133·08	77 0	—	—
54·0	135·49	78·25	0·50	3·3
55·0	138·11	79·5	—	—
57·9	38+145·40	83·0	—	—
	P = const.			
57·9	38+145·40	83·0	—	—
61 0	—	—	—	0·6
64·0	38+145·40	86 5	—	—
			Unloading of the muscle	[Elast. 0·26 × 10^{-3}]
64 0 + ϵ	38 + 0·0	48·0	—	—

Elasticity of the muscle during the interval (0—1″) = 0·20 × 10^{-3}

Elasticity of the muscle at the unloading = 0·26 × 10^{-3}

τ	$C_2\left(a - \frac{\Delta L}{\Delta P}\right)$	Lgn. C_1 (P ÷ Δ)	Diff.
12·6	− 0·968	− 0 968	0·0
26·0	− 0.830	− 0·829	+ 0·001
54·0	− 0·692	− 0·691	+ 0·001

$\Delta = 0·5$ $C_1 = 0·189 \times 10^{-3}$ $C_2 = -1333$

Length of the muscle, 10·5 cm.
Weight of the muscle, 34·2 grm.

TABLE XLIV.—*Experiment* 9 (December 6, 1900).

τ	P	L		Tonus	Plastic.
0·0	38 + 0·0	$0{\cdot}0 \times 10^{-3}$		—	—
0·5	—	—		$0{\cdot}26 \times 10^{-3}$	—
1·0	2·9	0·75		—	—
1·5	—	—		0·43	$4{\cdot}4 \times 10^{-3}$
2·0	5·8	2·0		—	—
2·5	—	—		0·52	4·5
3·0	8·7	3·5		—	—
3·5	—	—		0·52	4·4
4·0	11·6	5·0		—	—
4·6	—	—		—	4·4
5·2	15·08	7·0		—	—
6·2	17·98	9·0		0·69	4·7
7·2	20·88	11·0		—	—
9·5	—	—		—	4·4
11·6	33·63	19·25	0·69		
12·6	36·53	21·25		0·60	4·2
13·6	39·43	22·75	0·52		
25·0	72·49	41·5		—	—
26·0	75·39	43·0		0·52	3·7
27·0	78·29	44·5		—	—
33·0	—	—		—	3·5
39·0	113·08	61·75	0.52		
40·0	115·98	63·25		0·47	3·4
41·0	118·88	64·5	0·43		
45·0	—	—		0·42	—
48·7	38 + 141·21	74·0		—	—
	P = const.				
48·7	38 + 141·21	—		—	—
52·0	—	—		—	0·5
54·8	38 + 141·21	—		—	—
				Unloading of the muscle	$\left[\text{Elast. } 0{\cdot}27 \times 10^{-3}\right]$
$54{\cdot}8 + \epsilon$	38 + 0·0	39·0		—	—

Elasticity of the muscle during the interval (0—1″) $= 0{\cdot}26 \times 10^{-3}$

Elasticity of the muscle at the unloading $= 0{\cdot}27 \times 10^{-3}$

τ	$C_2 \left(a - \frac{\Delta L}{\Delta P}\right)$	Lgn. $C_1 (P - \Delta)$	Diff.
6·2	− 1·126	− 1·122	+ 0·004
12·6	− 0·979	− 0·986	− 0·007
26·0	− 0·849	− 0·848	+ 0·001
40·0	− 0·767	− 0·766	+ 0·001

$\Delta = 0{\cdot}5 \qquad C_1 = 0{\cdot}148 \times 10^{-3} \qquad C_2 = -1632.$

TABLE XLV.—*Experiment* 10 (April 6, 1902).

T	P	L	Tonus
0·0	50 + 0	$0{\cdot}0 \times 10^{-3}$	—
1·25	89	0·0	$0{\cdot}05 \times 10^{-3}$
3·75	267	12·9	0·05
6·25	445	19·3	—
6·5	463	25·8	—
9·0	641	38·7	0·07
11·5	819	51·6	—
11·75	836	51·6	—
14·25	1014	64·4	0·09
16·75	1192	83·8	—
20·0	1424	109·6	—
22·5	1602	128·9	0·11
25·0	1779	148·2	—
32·5	2313	212·7	—
35·0	2491	232·0	0·13
37·5	2669	257·7	—
41·0	2918	290·0	—
43·5	3096	341·6	0·25
46·0	3274	380·0	—
52·0	3701	470·5	—
54·5	3879	502·7	0·18
57·0	4057	534·9	—
65·5	4662	625·2	—
68·0	4840	644·5	0·11
70·5	5018	663·8	—
82·5	5872	721·8	—
85·0	6050	728·3	0·04
87·5	50 + 6228	734·7	—
	P = const.		
97·5	50 + 6228	734·7	—

TABLE XLVI.—*Experiment* 11 (March 16, 1902).

T	P	L	Tonus
0·0	50 + 0	$0{\cdot}0 \times 10^{-3}$	$0{\cdot}14 \times 10^{-3}$
1·25	92	12·9	
3·75	275	51·6	0·18
6·25	458	77·3	—
6·5	477	90·2	—
9·0	660	141·8	0·30
11·5	843	199·8	—
11·75	862	206·2	—
14·25	1045	270·7	0·39
16·75	1228	348·0	—
20·0	1467	425·4	—
22·5	1650	476·9	0·28
25·0	1833	528·5	—
25·5	1870	541·4	—
28·0	2053	580·1	0·21
30·5	2237	618·7	—
32·5	2383	644·5	—
35·0	2567	670·3	0·15
37·5	2750	696·1	—
41·0	3007	728·3	—
43·5	3190	741·2	0·07
46·0	3373	754·1	—
52·0	3813	773·4	—
54·5	3997	773·4	0·00
57·0	50 + 4180	773·4	—
	P = const.		
62·0	50 + 4180	773·4	—

TABLE XLVII.—*Experiment* 12 (April 6, 1902).

T	P	L	Tonus
0·0	50 + 0	0·0 × 10^{-3}	—
1·25	70	8·5	0·16 × 10^{-3}
3·75	210	34·2	0·24
6·25	350	68·4	—
6·5	364	77·0	—
9·0	504	128·3	0·40
11·5	643	188·2	—
11·75	657	196·7	—
14·25	797	273·7	0·55
16·75	937	350·7	—
20·0	1119	470·4	—
22·5	1259	564·4	0·73
25·0	1399	675·7	—
32·5	1818	1017·8	—
35·0	1958	1128·9	0·73
37·5	2098	1223·0	—
41·0	2294	1334·2	—
43·5	2434	1419·8	0·61
46·0	2574	1505·3	—
52·0	2909	1684·9	—
54·5	3049	1753·3	0·49
57·0	3189	1821·7	—
65·5	3665	2027·0	—
68·0	3805	2086·9	0·37
70·5	3944	2129·6	—
82·5	4616	2249·4	0·18* (?)
85·0	50 + 4756	2275·0	
	P = const.		
90·0	50 + 4756	2275·0	—

* The movement of the foot is checked in the malleolar joint

TABLE XLVIII.—*Experiment* 13 (March 9, 1902).

T	P	L	Tonus
0·0	50 + 0	0·0 × 10^{-3}	—
1·25	51	25·7	0·51 × 10^{3}
3·75	152	77·0	0·46
6·25	253	119·7	—
6·5	264	119·7	—
9·0	365	171·1	0·51
11·5	466	222·4	—
11·75	476	230·9	—
14·25	578	290·8	0·59
16·75	679	350·7	—
20·0	811	436·2	—
22·5	912	504·6	0·68
25·0	1013	573·0	—
32·5	1317	778·3	—
35·0	1419	855·3	0·76
37·5	1520	932·3	—
41·0	1662	1034·9	—
43·5	1763	1120·4	0·84
46·0	1865	1205·9	—
52·0	2108	1394·1	—
54·5	2209	1462·5	0·64
57·0	2311	1522·4	—
65·5	2655	1673·4	—
68·0	2757	1736·2	0·42
70·5	2858	1779·0	—
82·5	3344	1924·4	—
85·0	3446	1950·0	0·21
87·5	50 + 3547	1967·1	—
	P = const.		
90·0	50 + 3547	1967·1	—

TABLE XLIX.—*Experiment* 14 (November 29, 1899).

T		P	L	Tonus	Plastic.
0·0		1·9 + 0·0	$0{\cdot}00 \times 10^{-8}$	—	—
0·25		—	—	$2{\cdot}28 \times 10^{-8}$	—
0·5		0·51	1·07	—	—
0·75		—	—	2·36	$20{\cdot}60 \times 10^{-8}$
1·0		1·03	2·28	—	—
1·25		—	—	3·44	19·35
1·5		1·53	4·08	—	—
1·75		—	—	4·22	19·85
2·0		2·05	6·18	—	—
2·25		—	—	4·74	19·44
2·5		2·56	8·60	—	—
2·75		—	—	7·88	21·64
3·0		3·07	12·86	—	—
3·25		—	—	6·82	20·17
3·5		3·58	15·59	—	—
4·8		—	—	—	18·72
6·2		6.34	31·44	5·25	—
8·4	Stimulation of pretibial muscles				
10·0		10·30	52·94	—	—
10·5		—	—	5·22	22·71
11·0		11·25	58·32	—	—
11·5		—	—	5·22	16·32
12·0		12·28	68·69	—	—
12·4					
14·5		14·83	74·98	—	—
14·75		—	—	3·69	—
15·0		15·35	76·86	—	14·21
15·25		—	—	3·16	—
15·5		15·86	78·47	—	—
15·75		—	—	3·16	—
16·0		16·37	80·09	—	—
17·9	Stimulation of pretibial muscles				
18·0		18·40	85·73	—	—
18·5		—	—	4·17	14·20
19·0		19·43	90·08	—	—
19·5		—	—	3·65	13·53
20·0		20·46	93·79	—	—
21·4					
25·4		25·98	109·92	2·10	—
27·4		—	—	2·15	11·26
29·3		29·97	118·52	—	—
	P = const.				
29·3		1·9+29·97	118·52	—	—
29·6		—	—	—	0·38
30·0		—	118·79	—	—
31·0		—	—	—	0·27
32·0		—	119·33	—	—
33·0		1·9+29·97	119·60 (?)	—	—
	Unloading of the muscle.				
33·0 + ε		1·9 + 0·0	(?)	—	—

TABLE L.—*Experiment* 15 (November 26, 1899).

τ		P	L	Tonus	Plastic.
0·0		1·9 + 0·00	0·00 × 10^{-3}	—	—
0·5		0·54	1·61	3·45 × 10^{-3}	—
0·75		—	—	3·91	16·77 × 10^{-3}
1·0		1·09	3·76	—	—
1·25		—	—	5·48	—
1·5		1·63	6·72	—	17·98
1·75		—	—	7·88	—
2·0		2·18	10·75	—	—
2·25		—	—	6·97	17·61
2·5		2·72	14·51	—	—
2·75		—	—	6·59	[illegible]·21
3·0		3·27	18·14	—	—
3·3	Stimulation of pretibial muscle	—	—	—	—
3·5		3·81	23·11	—	—
3·75		—	—	6·97	16·80
4·0		4·35	26·87	—	—
4·25		—	—	7·47	17·31
4·5		4·90	30 91	—	—
4·75		—	—	7·97	16·45
5·0		5·44	34·67	—	—
5·5		—	—	6·90	16·29
6·0		6·53	42·19	—	—
6·5		—	—	5·92	14·96
7·0		7·62	48·64	—	—
7·5		—	—	6·65	15·55
8·0		8·71	55·90	—	—
8·25		—	—	5·97	15·68
8·5		9·25	59·12	—	—
8·6		—	—	—	—
11·6		12·63	77·13	—	—
12·1		—	—	4·68	12·68
12·6		13·72	82·24	—	—
13·1		—	—	4·68	12·57
13·6		14·81	87·34	—	—
22·0		23·95	120·94	—	—
22·5		—	—	3·70	10·68
23·0		25·04	124·97	—	—
23·5		—	—	3·70	10 62
24·0		26·13	129·00	—	—
24·4		—	—	3·70	—
24·8		27·0	132·22	—	—
	P = const.				
24·8		1·9 + 27·0	132·22	—	—
26·0		—	132·76	—	—
26·5		—	—	—	0·54
27·0		—	133·30	—	—
27·3		—	—	—	0·45
27·6		1·9 + 27·0	133·57	—	—
			Unloading of the muscle		[Elastic 3·58 × 10^{-3}]
27·6 + ϵ		1·9 + 0·00	36·82	—	—
28·5		—	10·75 (?)	—	—
29·0		—	—	—	− 2·15
29·5		—	8·60	—	—
30·5		—	—	—	− 1·07
31·5		—	6·45	—	—
32·0		—	—	—	− 0·44
32·5		1·9 + 0·00	5·91	—	—

TABLE LI.—*Experiment* 16 (October 25, 1909).

τ		P	L	Tonus
0 0		1·96 + 0·0	$0{\cdot}00 \times 10^{-3}$	—
1·0		1·01	1·65	$+\ 2{\cdot}86 \times 10^{-3}$
2·0		2·02	5·77	—
2·5		—	—	+ 5·71
3·0		3·03	11·54	—
3·5		—	—	+ 7·07
4·0		4·04	18·68	—
4·5		—	—	+ 7·07
5·0		5·05	25·83	—
5·4	Stimulation of pretibial muscles	—	—	+ 6·04
5·5		5·55	28·85	—
5·55		—	—	+10·00
5·6		5·66	29·95	—
5·65		—	—	+ 5·00
5·7		5·76	30·50	—
5·75		—	—	+ 5·50
5·8		5·86	31·05	—
5·85		—	—	+ 2·70
5 9		5·96	31·32	—
5·95		—	—	+ 0·60
6·0		6·06	31·38	—
6·05		—	—	− 3·30
6·1		6·16	31·05	—
6·15		—	—	− 8·30
6·2		6·26	30·22	—
6·25		—	—	0·00
6·3		6·36	30·22	—
6·35		—	—	+ 2·80
6·4		6·46	30·58	—
6·45		—	—	+ 5·50
6·5		6·56	31·05	—
6 55		—	—	+ 2·70
6·6		6·67	31·32	—
6·65		—	—	0·00
6·7		6·77	31·32	—
6·75		—	—	0·00
6·8		6·87	31·32	—
6·85		—	—	+ 2·80
6·9		6·97	31·60	—
6·95		—	—	+ 5·50
7·0		7·07	32·15	—
7·05		—	—	+ 8·20
7·10		7·17	32·97	—
7·15		—	—	+11·00
7·2		7·27	34·07	—
7·25		—	—	+13 70
7·3		7·37	35·44	—
7·35		—	—	+13·80
7·4		7·47	36·82	—
7·45		—	—	+10·00
7·50		7·57	37·92	—
7·55		—	—	+10·90
7·60		7·68	39·01	—
7·65		—	—	+11·00
7·70		7·78	40·11	—
7·75		—	—	+ 8·30
7·80		7·88	40·94	—
7·85		—	—	+ 5·50
7·9		7·98	41·49	—
7·95		—	—	+ 5·50
8·0		8·08	42·04	—
8·5		—	—	+ 5 20
9·0		9·09	47·26	—
9·5		—	—	+ 4·60
10·0		10·10	51·98	—
10·5		—	—	+ 3·80
11·0		11·11	55·77	—
11·5		—	—	+ 3 50
12·0		12·12	59·35	—
12·1		12 22	—	—

[THESE TABLES SHOULD BE READ IN CONJUNCTION WITH CHAPTER III.]

THE ISOTONIC CONTRACTION.

TABLE LII.—*Experiment* 6.—TIGERSTEDT and WILLHARD.

No.	P	$C_1(P-\Delta)$	L. obs.	L. calc.	Const.
1	65	6·9	1·8	2·2	$\Delta = 62$ $C_1 = 2·30$ $C_2 = 0·90$
2	70	18·4	4·4	4·0	
3	75	29 9	5·4	5·2	
4	80	41·5	6·1	6·0	
5	85	53·0	6·6	6·45	
6	90	64·5	6·65	6·7	
7	95	76·0	6·75	6·75	
8	95	76·0	6·7	6·7	$\Delta = 60$ $C_1 = 2$ 17 $C_2 = 0·96$
9	90	65·1	6·6	6·65	
10	85	54·3	6·2	6·4	
11	80	43·4	6·15	6·0	
12	75	32 6	5·0	5·35	
13	70	21·7	4·3	4·35	
14	65	10·9	3·1	2·9	

TABLE LIII.—*Experiment* 28.—TIGERSTEDT and WILLHARD.

No.	P	$C_1(P-\Delta)$	L. obs.	L. calc.	Const.
1	40	0 34	0·95	0·3	$\Delta = 39·9$ $C_1 = 3·44$ $C_2 = 0·39$
2	42	7·2	2·6	3·4	
3	44	14 1	5·15	5 3	
4	46	20·5	6·65	6·65	
5	48	27·9	7·8	7·8	
6	50	34 7	8·9	8·65	
7	52	41·6	9·45	9·3	
8	54	48·5	9·8	9·8	
9	56	55·4	10·15	10·15	
10	58	62·3	10·4	10 35	
11	60	69·1	10·45	10·45	
12	62	76·0	10·5	10·5	
13	64	82·9	10·6	10·45	
14	66	—	10·65	—	
15	68	—	10·65	—	
16	70	—	10·7	—	
17	75	—	10·7	—	
18	80	—	10·7	—	
19	85	—	10·75	—	
20	85	—	10·8	—	$\Delta = 40·6$ $C_1 = 3·55$ $C_2 = 0·36$
21	80	—	10·85	—	
22	75	—	10·85	—	
23	70	—	10·85	—	
24	68	—	10·9	—	
25	66	90·12	10·85	10·7	
26	64	83·1	10·9	10·87	
27	62	76·0	10 9	10·9	
28	60	68·6	10·9	10·9	
29	58	61·5	11·05	10·8	
30	56	54·4	10·75	10·5	
31	54	47·4	10·4	10·1	
32	52	40·3	10·0	9·55	
33	50	33·2	9·1	8·8	
34	48	26·2	8·0	7·9	
35	46	19·1	6 65	6·65	
36	44	12 0	4·9	4·95	
37	42	4·9	0·0	2·7	
38	40	0·0	0·0	0·0	

TABLE LIV.—*Experiment* 38.—TIGERSTEDT and WILLHARD.

No.	P	$C_1(P-\Delta)$	L. obs.	L. calc.	Const.
1	55	19·2	5·8	5·85	$\Delta = 48·2$ $C_1 = 2·84$ $C_2 = 0·51$
2	60	33·4	7·8	7 8	
3	65	47·6	8·8	8·95	
4	70	61·8	9·4	9·5	
5	75	76·0	9·65	9·65	

TABLE LV.—*Experiment* 32A.—TIGERSTEDT and WILLHARD.

No.	P	$C_1(P-\Delta)$	L. obs.	L. calc.	Const.
1	4·1	5·7	0 5	3·65	$\Delta = 1·5$ $C_1 = 2·18$ $C_2 = 0·48$
2	7·7	13·5	6·65	6·65	
3	10·9	20·5	8·85	8·55	
4	14·4	28·1	10·2	10·1	
5	18·1	36·1	11·0	11·3	
6	21·6	43·8	12·0	12·15	
7	25·2	51·6	12·2	12·8	
8	28·8	59·4	13·0	13·2	
9	32·8	68·1	13·4	13·4	
10	36·4	76·0	13·5	13·5	
11	36 4	76·0	14·4	13·8	$\Delta = 0$ 5 $C_1 = 2·12$ $C_2 = 0·50$
12	32·8	68·4	13·75	13·75	
13	28·8	59 9	12·9	13·55	
14	25·2	52·3	12·65	13·1	
15	21·6	44.7	12·4	12·5	
16	18·1	37·3	11·4	11·65	
17	14·4	29·4	10·8	10·5	
18	10·9	22·0	9·7	9·05	
19	7·7	15 2	7·1	7 3	
20	4·1	7·6	1·15	4·6	

TABLE LVI.—*Experiment* 2.—Von Kries.

No.	P	$C_1(P-\Delta)$	L. obs.	L. calc.	Const.	No.	P	$C_1(P-\Delta)$	L. obs.	L. calc.	Const.
1	17	0·0	3·0	0·0	$\Delta = 17$ $C_1 = 5·07$ $C_2 = 0·25$	8	32	76·0	11·1	11·1	$\Delta = 17·25$ $C_1 = 5·15$ $C_2 = 0·24$
2	18	5·1	3·0	2·75		9	28	55·4	10·7	10·75	
3	20	15·2	5·5	5 8		10	26	45·1	10·1	10·1	
4	22	25·3	6·0	7·75		11	24	34·8	8·8	9·15	
5	24	35·5	9·0	9·1		12	22	24·5	7·0	7·7	
6	26	45·6	10·0	10·0		13	20	14·2	5·5	5·6	
7	28	55·7	10·2	10·5		14	18	3·9	2·6	2·3	
8	32	76·0	11·1	10·95		15	17	0·0	2·6	0·0	

TABLE LVII.—*Experiment* 3.—Cybulski and Zanietowski.

Isotonic Series.

No.	P coulombs	L. obs.	No.	P coulombs	L. obs.
10	$25·4 \times 10^{-10}$	29·30	19	33·4	32·18
11	27 4	29·64	20	33·5	32·54
12	29 6	29·98	21	33·6	32·81
13	30·8	30·30	22	33·7	33 07
14	32·6	30·60	23	33·8	33·43
15	33·0	30·96	24	33 9	33·74
16	33·1	31·29	25	34·0	34·01
17	33·2	31·60	26	34·1	34·32
18	33·3	31·92	27	34·2	34·41

TABLE LVIII.—*Experiment* 2.—Cybulski and Zanietowski.

Isotonic Series.

No.	P coulombs	$C_1(P-\Delta)$	L. obs.	L. calc.	Const.
1	$26·3 \times 10^{-11}$	0·0	1·0	0·0	$C_1 = 14·34$ $\Delta = 26·6$ $C_2 = 0·034$,
2	26·80	2·86	3·8	4·65	
3	27·17	8·17	8·6	10·05	
4	27·53	13·34	14·3	13·95	
5	27·90	18·64	16·2	17·0	
6	28·30	24·38	19·6	19·8	
7	28·60	28·68	20·2	21·5	
8	28·96	33·84	22·4	23·15	
9	29·30	38·72	23 0	24·55	
10	29·60	43·02	26·2	25·55	
11	29·98	48·57	26·4	26·55	
12	30·30	53·16	26·5	27·2	
13	30·60	57·36	28·0	27·75	
14	30·96	62·62	28·2	28·15	
15	31·29	67·35	28·3	28·4	
16	31·60	71·70	28·4	28·45	
17	31·90	76·00	28·5	28·5	
18	32·18	80·3	28·6	28·4	
19	32·50	84·6	28·7	28·3	
20	32·80	—	28·8	—	
21	33·07	—	28·9	—	
22	33·43	—	29·0	—	
23	33·70	—	29·2	—	
24	34·01	—	29·2	—	

TABLE LIX.—*Experiment* 6.—CYBULSKI and ZANIETOWSKI.

ISOTONIC SERIES.

(A.)

No.	P coulombs	$C_1\ (P-\Delta)$	L. obs.	L. calc.	Const.
1	10×10^{-10}	0·0	0·25	0·0	$\Delta=11{\cdot}0$, $C_1=6{\cdot}91$, $C_2=0{\cdot}078$
2	10·95	0·0	4·0	0·0	
3	11·85	5·87	6·1	7·25	
4	12·65	11·40	11·6	11·5	
5	13·40	16·58	14·8	14·6	
6	14·15	21·76	16·7	17·0	
7	14·85	26·60	20·3	18·95	
8	15·50	31·09	21·5	20·4	
9	16·10	35·24	21·6	21·6	
10	16·75	39·73	22·6	22·65	
11	17·30	43·53	22·9	23·45	
12	17·90	47·67	24·3	24·15	
13	18·45	51·47	25·0	24·7	
14	18·95	54·93	25·2	25·1	
15	19 50	58·73	25·4	25·4	
16	20·00	62·18	25·6	25·7	
17	20·50	65·64	25·8	25·9	
18	20·95	68·74	26·0	26·0	

(B.)

No.	P coulombs	$C_1\ (P-\Delta)$	L. obs.	L. calc.	Const.
1	$14{\cdot}15 \times 10^{-10}$	0·0	0·25	0·0	$\Delta=14{\cdot}8$, $C_1=8{\cdot}0$, $C_2=0{\cdot}064$
2	14·85	0·0	0·25	0·0	
3	15 50	8·0	7·6	9·5	
4	16·10	12·8	12·3	13·0	
5	16·75	18·0	16·5	16·05	
6	17·30	22·4	17·7	18·15	
7	17·90	27·2	19·7	20·0	
8	18·45	31·6	21·0	21·6	
9	18·95	35·6	22·4	22·8	
10	19·50	40·0	24·0	23·9	
11	20·00	44·0	24·9	24·75	
12	20·50	48·0	25·1	25·4	
13	20·95	51·6	26·0	26·0	

TABLE LX.—*Experiment of* CYBULSKI and ZANIETOWSKI.

ISOTONIC SERIES.

Curve I (*Twitch*).

No.	P coulombs	$C_1\ (P-\Delta)$	L. obs.	L. calc.	Const.
1	$37{\cdot}41 \times 10^{-10}$	0·0	0·25	0·0	$\Delta=39{\cdot}8$, $C_1=14{\cdot}62$, $C_2=0{\cdot}106$
3	38·73	0·0	0·75	0·0	
5	40·00	2·92	1·5	1·5	
7	41·28	20·90	4·0	5·75	
9	42·43	38·44	7·5	7·7	
11	44·72	71·90	9·0	9·0	

Curve II (*Tetani*).

No.	P coulombs	$C_1\ (P-\Delta)$	L. obs.	L. calc.	Const.
2	$37{\cdot}41 \times 10^{-10}$	0·0	0·75	0·0	$\Delta=37{\cdot}65$, $C_1=6{\cdot}13$, $C_1=0{\cdot}085$
4	38·73	6·62	8·0	8·1	
6	40·00	14·40	13·5	13·6	
8	41·28	21·95	16·5	17·4	
10	42·43	29·30	20·0	20·2	
12	44·72	43·34	24·0	23·9	

TABLE LXI.—*Experiment of* WALLER.

No.	P	$C_1(P-\Delta)$	L. obs.	L. calc.	Const.
1	1·52	0·0	0·0	0·0	$\Delta=1{\cdot}59$, $C_1=120{\cdot}6$, $C_2=0{\cdot}0085$
2	1·65	7·24	4·0	4·1	
3	1·79	24·13	9·0	8·7	
4	1·97	45·84	12·0	11·6	
5	2·13	65·15	12·5	12·5	
6	2·32	88·07	12·5	12·4	

TABLE LXII.—*Experiment* 6.—KEITH LUCAS.

No.	P Defl. galv.	$C_1(P-\Delta)$	L. obs.	L. calc.	Const.
1	39·45	16·7	0·0	3·84	$\Delta = 37·75$ $C_1 = 9·81 \quad C_2 = 0·208$
2	39·85	20·6	3·36	4·34	
3	40·25	24·5	4 96	4·77	
4	40·65	28·4	5·12	5·14	
5	41·05	32·4	5·44	5·47	
6	41·45	36·3	5·46	5·74	
7	41·85	40·2	5·47	5·98	
8	42·25	41·1	6·28	6·18	
9	42·65	48·1	6·37	6·35	
10	43·05	52·0	6·39	6·49	
11	43·50	56·4	6·10	6·63	
12	44·00	61·3	6·72	6·73	
13	44·50	66·2	6·74	6·81	
14	45·00	71·1	6·76	6·83	
15	45·50	76·0	6·85	6·85	
16	46·00	80·9	6·88	6·82	
17	46·50	—	6·85	—	
18	47 00	—	6·86	—	
19	52·00	—	6·88	—	
20	58·00	—	6·89	—	
21	76·25	—	6·88	—	
22	100·25	—	6·87	—	
23	140 00	—	6·90	—	
24	200·00	—	6·88	—	

TABLE LXIII.—*Experiment* 11.—GILDEMEISTER.

CURVE I.

No.	P	$C_1(P-\Delta)$	L. obs.	L. calc.	Const.
1	15	14·9	8·75 9·25	10·0	$\Delta = 12·8$ $C_1 = 6·78 \quad C_2 = 0·108$
3	16	21·7	12·25 12·50	12·45	
5	17	28·5	15·25 15·00	14·35	
7	18	35·3	16·00 16·00	15·80	
9	19	42·1	16·75 17·00	16·95	
11	20	48·8	17 75 17·50	17·80	
13	25	82·8	19·00 19 25	19·00	
15	30	116·7	18 75 18·75	16·42	
17	40	—	19 00 19·00	—	
19	50	—	19·00 19·25	—	
21	60	—	19·00 19·00	—	
23	70	—	19·00 19·00	—	
25	80	—	19 00 19·25	—	
27	90	—	19·00 19·00	—	
29	100	—	19·00 19·00	—	

CURVE II.

No.	P	$C_1(P-\Delta)$	L. obs.	L. calc.	Const.
2	15	0·6	0·0	1·15	$\Delta = 14$ $C_1 = 0·56 \quad C_2 = 0·967$
4	16	1·1	0·0	2·02	
6	17	1·7	0·0	2·81	
8	18	2·2	0·0	3·52	
10	19	2·8	0·0	4·16	
12	20	3·3	0·0	4·79	
14	25	6·1	0·0	7·49	
16	30	8·9	0·0	11·36	
18	40	14·5	11·00 11·25	13·3	
20	50	20·1	16·00 16·25	16·15	
22	60	25·7	19 00 19·00	18·45	
24	70	31·3	20·5 20·5	20·30	
26	80	36·9	21·25 21 00	21·80	
28	90	42·5	22·75 23·00	23·00	
30	100	48·1	24·00 24·00	24·00	

TABLE LXIV.—*Experiment* 2.—MAY.

No.	P	$C_1(P-\Delta)$	L. obs.	L. calc.	Const.
1	20	7·86	17·5	14·2	$C_1 = 0·52$ $\Delta = 5$ $C_2 = 0·65$
2	35	15·72	22·5	22·35	
3	50	23·58	27·0	28·15	
4	60	28·25	30·5	30·85	
5	70	34·07	33·0	33·7	
6	80	39·31	36·0	35·8	
7	90	44·55	37·0	37·5	
8	100	49·79	38·0	38·8	
9	110	55·03	38·5	39·65	
10	120	60·27	40·5	40·4	
11	130	65·51	40·5	41·0	
12	140	70·75	41·25	41·2	
13	150	76·00	41·3	41·3	
14	160	81·24	40·5	41·0	
15	170	86·48	40·0	40·6	
16	180	91·72	40·0	40·1	
17	190	96·96	39·5	39·5	
18	200	102·20	38·0	38·5	

TABLE LXV.—*Experiment* 3.—MAY.

No.	P	$C_1(P-\Delta)$	L. obs.	L. calc.	Const.
1	20	13·79	9·5	8·1	$C_1 = 0·71$ $\Delta = 0·5$ $C_2 = 1·23$
2	35	24·75	11·75	11·25	
3	50	35·35	13·75	13·35	
4	65	45·95	14·75	14·75	
5	80	56·56	15·5	15·5	
6	100	70·70	15·75	16·0	
7	125	88·37	(?)	15·75	
8	150	106·05	14·75	14·75	
9	175	123·72	13·75	13·10	
10	200	141·40	11·5	11·20	

TABLE LXVI.—*Experiment* 4.—MAY.

No.	P	$C_1(P-\Delta)$	L. obs.	L. calc.	Const.
1	20	8·14	28·0	20·8	$C_1 = 0·45$ $\Delta = 2$ $C_2 = 0·52$
2	35	14·93	31·0	30·9	
3	50	21·72	38·5	38·5	
4	60	26·24	43·0	42·55	
5	70	30·76	46·25	46·0	
6	80	35·29	49 25	48·9	
7	90	39·81	51·50	51·4	
8	100	44·34	53·0	53·35	
9	110	48·86	54·5	55·1	
10	120	53·38	55·25	56·3	
11	130	57·91	56·25	57·4	
12	140	62·43	57·25	58·2	
13	150	66·96	58·50	58·75	
14	160	71·48	58·75	58·8	
15	170	76·00	59·0	59·0	
16	180	80·53	58·5	58·75	
17	190	85·05	58·0	58·15	
18	200	89·58	57·75	57·85	

UNMYELINATED NERVE-FIBRES AS CONDUCTORS OF PROTOPATHIC SENSATION.

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THE object of this paper is to bring together a number of scattered observations on the structure and function of the afferent fibres of the cerebrospinal nerves, and to formulate on the basis of these observations a working hypothesis, which should greatly facilitate further investigation along these lines. As yet we do not know how the sensations of touch, pain, and temperature are differentiated and how the underlying afferent impulses are propagated along the peripheral nerves.

PROTOPATHIC NERVE-FIBRES.

An important advance was made by Head and his associates (1905, 1906, 1908) when they showed that cutaneous sensations could be separated into two groups, to which they applied the terms "protopathic" and "epicritic." Under the term "protopathic," Head groups, in addition to pain, the temperature sensations aroused by objects under 22° C. or over 40° C. This group is characterized by a "peculiar tingling quality," by radiation into other parts than those stimulated, and by failure of the subject to localize accurately the point stimulated. Under the term "epicritic" he groups sensibility to light touch, temperature sensations derived from objects between about 22° C. and 40° C.,[1] and discrimination of the two compass points. Sensations of this kind are all accurately localized. It would take too much space to tell in detail how these two types of sensation were separated from each other. Obviously such a distinction could not be made by a study of the normal skin. It was found, however, that after lesions of the dorsal roots areas of pure epicritic sensation appeared. Such a cutaneous area was sensitive to light touch and to medium degrees of temperature, but insensitive to pain and to the more extreme degrees of temperature. On the other hand, when the median nerve was cut a cutaneous field became outlined on the palm of the hand, in which only protopathic sensations were experienced. Here the skin was sensitive to pain and to the extreme degrees of temperature, but insensitive to light touch and the intermediate degrees of temperature. All sensations from such an area were poorly localized and had a peculiar tingling quality. Head and his associates studied a very large series of cases with nerve lesions and found many such areas of dissociated sensation.

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[1] These are the approximate limits as determined in clinical work on divided peripheral nerves. In the experiment recorded by Rivers and Head these limits could be more accurately determined and were found to lie between 25° or 26° C., and 37° to 38° C.—[ED.]

I have myself had an opportunity of studying in one of my students a partially anæsthetic area in the palm of the hand, resulting from the division of the median nerve, and have found it to possess all the characteristics of a protopathic area. There is complete loss of sensibility to light touch over the entire area of distribution of the median nerve. But over a considerable portion of this area the skin is acutely sensitive to the prick of a pin. A test-tube filled with ice water was felt as cold and one filled with water at 50° C. as hot, but test-tubes with water at 22° C. and 40° C. were not distinguishable from each other. Sensations from this area were not well localized, and had a peculiarly unpleasant character. In other words, the area possessed sensation of a typically protopathic character.

It is maintained by Head that each of his two sensory groups depends on a separate, anatomically distinct, set of nerve-fibres. He presents good and convincing reasons for this belief, but space does not permit us to repeat the argument here. We wish only to call attention to a fact which might easily be overlooked in reading the original articles. In areas of partial anæsthesia the residual sensation may be either protopathic or epicritic. If the only form of residual sensation were protopathic, one might assume that it depended only on a decreased density of innervation. It might easily be that light touch required a denser innervation for its perception than pain. But the reverse form of partial anæsthesia also occurs (and numerous examples of it are given by Head) in which epicritic sensation persists over an area devoid of protopathic sensation. It is not conceivable that a simple decreased density of innervation should in the one case give rise to a loss of light touch with pain persisting, and in another case cause a complete loss of pain sense while light touch remains normal. Furthermore, these areas of pure epicritic sensation are sensitive to temperature between about 22° C. and 40° C., but insensitive to the more extreme degrees of temperature. This is clearly not a case of lowered sensibility due to decreased density of innervation. It seems clear to me that these facts can be explained only on Head's assumption that there are two kinds of afferent nerve-fibres, which differ slightly in their anatomical distribution.

According to Head the unit of distribution of protopathic fibres is the dorsal root, each root having a sharply outlined area of skin which it supplies with them. The epicritic fibres of adjacent roots are intermingled in their cutaneous distribution. Section of one or more dorsal roots deprives a sharply circumscribed area of skin of its protopathic fibres, while epicritic fibres from adjacent roots run into this area, endowing more or less of the skin near its border with pure epicritic sensation. Here light touch is felt, but not pain, warm and cold objects are discriminated, but hot and cold objects give rise to no temperature sensation. In the same way the peripheral nerve is the unit of epicritic sensation. The epicritic fibres of the ulnar nerve are limited to the area of skin outlined by anatomists as representing the cutaneous distribution of that nerve; but the protopathic fibres of the ulnar run long distances in the subcutaneous plexuses into the areas belonging to adjacent nerves. When the median nerve is cut epicritic sensation is lost over the entire area ordinarily assigned by anatomists to that nerve, but protopathic fibres from the ulnar nerve run into this area endowing a considerable extent of the skin near the border of the area with pure protopathic sensation. Here pain is felt but not touch. Hot and cold objects are distinguished, but warm and cool objects give rise to no temperature sensation.

Facts which are otherwise inexplicable are thus readily understood on the assumption of two kinds of nerve-fibres which vary slightly in their anatomical distribution. This assumption acquires still greater significance in view of the recent demonstration that there are two kinds of afferent cerebrospinal nerve-fibres which differ both in structure and distribution. Until recently we were acquainted with only one of these, the myelinated fibres. But Ranson has recently shown that there are great numbers of unmyelinated afferent fibres that had previously been overlooked. Can it be that one kind, say the myelinated, is responsible for epicritic sensation and the other, the unmyelinated, for protopathic sensation? In the résumé which follows we shall see that there is the most striking parallelism between what is known of the protopathic fibres and what has recently been determined in regard to the unmyelinated afferent nerve-fibres.

UNMYELINATED AFFERENT NERVE-FIBRES.

Ranson (1912) modified the Cajal silver technique in such a way as to produce a differential stain of unmyelinated fibres, and applied to the method the term "pyridine silver technique." When the spinal nerves are stained by this method they are seen to contain great numbers of unmyelinated fibres. These are, in fact, more numerous than those which are myelinated. It is possible to trace them centrally and to show that they arise from the small cells of the spinal ganglia. These small spinal ganglion cells possess single axons which divide dichotomously into fibres running into the peripheral nerves on the one hand and into the dorsal roots on the other. These axons and their branches remain unmyelinated throughout their course. Fibres of this kind are also present in the cranial nerves, particularly in the vagus, where they can be demonstrated with the greatest ease (Chase and Ranson, 1914, Ranson, 1914). The two kinds of nerve-fibres seem to be sharply separately from each other with few or no transition forms in the shape of partially myelinated fibres. That the distinction between the two is a fundamental one is evidenced by the fact that they are present in the same relative proportion and distributed in the same way in the vagus nerve of a reptile (the snapping turtle) as in that of a mammal (the dog) (Ranson, 1915). It would be strange if such a sharp division of afferent nerve-fibres into two kinds according to structure did not run parallel to the equally sharp division of these fibres according to function. We should, therefore, expect to be able to assign protopathic sensation to one and epicritic to the other. In fact, we shall see that the protopathic and unmyelinated fibres have a great many things in common.

Distribution of the afferent unmyelinated fibres.—The unmyelinated fibres of spinal nerves are distributed chiefly to the skin, but some go also with the muscular branches of the nerves. On the assumption that fibres of this kind mediate protopathic sensation those going to the deeper structure would account for the sense of pain in the muscles, joints and other deeply situated parts. The assumption that the fibres carrying cutaneous pain belong to a different class from those carrying pain from the muscles and joints is a weakness in the theory as presented by Head which we are now able to avoid.

The unmyelinated fibres are not distributed equally in all cutaneous nerves. The median nerve at the wrist contains an absolutely and relatively much greater number of myelinated nerve-fibres than does the medial cutaneous nerve of the forearm. The former is distributed to the skin of the fingers which possesses a high order of epicritic sensation, and it is composed predominantly of myelinated fibres; the latter is distributed to an area whose epicritic sensation is of a lower order, and it contains correspondingly fewer myelinated fibres so that the unmyelinated fibres greatly predominate. The lateral femoral cutaneous nerve is another instance of a nerve running to an area of low epicritic sensibility and containing relatively few myelinated fibres. There seems, therefore, to be a direct relation between the sharpness of epicritic sensation and the proportion of myelinated fibres in the associated nerve.

Rate of regeneration.—As yet we have no information regarding the relative rate of regeneration of the myelinated and unmyelinated afferent nerve-fibres. Head has shown, however, that after division and suture of a nerve protopathic sensation returns to the area supplied by that nerve much more quickly than does epicritic sensation. Protopathic sensation may be completely restored over the entire area supplied by the regenerating nerve for months before epicritic sensation begins to return. Now it is a significant fact that exactly at the time at which protopathic sensation is restored innervation returns to the sweat glands and blood-vessels. This means a regeneration of the vasomotor and secretory fibres, which are post-ganglionic autonomic (or sympathetic) fibres. These are known to be unmyelinated. On the assumption that the protopathic fibres are also unmyelinated the fact that these two groups of fibres complete their regeneration at the same time would be easily understood.

Visceral sensation.—In addition to sensations of movement and pressure the viscera give rise to sensations of a protopathic character. Sensations of movement and pressure are attributed by Head to the large myelinated visceral afferent fibres which run through the sympathetic system to end in pacinian corpuscles. The stomach and its mucosa are insensitive to touch. While under ordinary conditions sensations from the stomach are not recognized in consciousness, yet it is a matter of common experience that under certain conditions pain may be felt in that viscus. Carlson (1914) has shown that the mucosa of the stomach is sensitive to temperatures of 10° and 50° C., but insensitive to the intermediate degrees. He concludes that it is endowed with protopathic temperature sensation. In the same way Head showed that the colon was sensitive to temperatures in the protopathic range. The evidence, therefore, supports Head's contention that the viscera possess protopathic but not epicritic sensibility.

Now it has been shown that the afferent fibres of the vagus coming from the stomach are chiefly unmyelinated. The vagus nerve, as it pierces the diaphragm, is practically an unmyelinated nerve (Chase and Ranson, 1914). It contains relatively fewer myelinated fibres than many sympathetic nerves, but is solidly packed with unmyelinated axons. Molhant (1913) has shown that about one-quarter of the cells of the nodose ganglion give rise to fibres running to the stomach. This is an enormous number of cells, only a small part of which could be accounted for by the few myelinated fibres in the gastric rami, even if one overlooked the fact that at least some of these are probably visceral efferent fibres. It is therefore clear that the main sensory innervation of the stomach is with unmyelinated fibres, and that, here, a very close parallel exists between the distribution of these fibres and those mediating protopathic sensation.

Course of the afferent fibres in the spinal cord.—It is well known that the afferent impulses underlying sensations of pain and temperature pass through the grey matter and cross to the opposite side of the cord at or near the level at which they reach it. Head (1906) has shown that this is true for temperature sensation of the epicritic as well as of the protopathic order. According to him the other elements of the epicritic group (touch, tactile discrimination, and tactile localization) are carried upward on the same side of the cord in the posterior funiculus for varying distances before ending in the grey matter. He maintains that the tactile impulses coming in along a given root do not cross to the opposite side of the cord all at once, but that they ascend in the posterior columns for varying distances. The crossing at varying levels of impulses coming in by a single root gives rise to a double pathway for touch, uncrossed fibres of the first order paralleling crossed fibres of the second order for a certain number of segments. This double path no doubt accounts for the conflicting observations on the conduction of tactile impulses which are found in the literature.

The facts that have been ascertained regarding the intraspinal course of the unmyelinated fibres is in complete accord with the view that they are the conductors of protopathic afferent impulses. As a dorsal root enters the spinal cord the two kinds of fibres separate, the unmyelinated turn laterally into Lissauer's tract while the myelinated run on into the posterior funiculus (Ranson, 1913, 1914). Few, if any, unmyelinated fibres enter that funiculus, but a few fine myelinated fibres run into the tract of Lissauer. This consists chiefly of unmyelinated axons scattered among which are a few fine myelinated fibres. From the level at which they enter the cord, the fibres ascend or descend in this tract for a very short distance not exceeding one or two segments. The substantia gelatinosa seems to be the sensory nucleus associated with this tract.

The unmyelinated fibres, then, enter the grey matter at or near the level at which they enter the cord. In this they are again in exact agreement with the fibres conveying protopathic sensation. The myelinated fibres, which alone enter the posterior funiculus, correspond in their intramedullary course to the fibres carrying light touch, tactile discrimination and tactile localization, since, according to Head, these ascend for longer or shorter distances in this funiculus before entering the grey matter. As to the temperature sensation in the epicritic range, they are probably conveyed by the fine myelinated dorsal root-fibres that run with the unmyelinated ones into the tract of Lissauer. It is thus apparent that we have at hand data sufficient to explain the intramedullary course of the protopathic and epicritic sensation in terms of the demonstrated intramedullary course of the myelinated and unmyelinated fibres.

So far as the evidence goes the theory that protopathic sensation is conveyed by unmyelinated fibres seems to be well supported. A number of methods of testing this hypothesis have occurred to us and experiments directed to this end are now under way. Some of these have progressed far enough to give evidence of value.

The function of the tract of Lissauer.—Experiments on the spinal cord of the cat (Ranson and von Hess, 1915) have shown that the tract of Lissauer and the substantia gelatinosa Rolandi are at least closely associated with the pain reception and conduction apparatus. It was found that while bilateral destruction of the tract of Lissauer and the substantia gelatinosa at the level of the first lumbar segment of the cat's cord did not interfere in any way with the perception of pain in the hind limbs, it entirely eliminated the pressor vasomotor reflex from stimulation of the sciatic nerve. Now the vasomotor reflexes are distinctly protopathic in that they are produced almost exclusively by pain and temperature sensation. The evidence, presented in that paper, showed that the tract of Lissauer and the substantia gelatinosa formed a path for conduction of the afferent impulses involved in the reflex vaso-constriction due to painful sciatic stimulation. It seemed probable to us that the tract of Lissauer and the substantia gelatinosa Rolandi form an apparatus for the reception and intersegmental conduction of painful afferent impulses. Some impulses from this apparatus passing over to the spinothalamic tract would reach the cortex and find expression as conscious pain, while other impulses received in this apparatus would ascend and descend within it, producing pain reflexes. So far as the evidence goes this work favours the theory that the unmyelinated fibres conduct protopathic sensation in that it shows that the portion of the cord in which these fibres run and terminate forms part of a protopathic reflex arc.

We hope to obtain more direct evidence from two lines of investigation which we are about to undertake. The glans penis is a region possessing only deep and protopathic sensibility, and if the conception presented in this paper is correct the nerve-fibres distributed to the skin of this region should be for the most part unmyelinated. Again, by experimental work on animals, it should be possible to find hyperæsthetic areas at the borders of anæsthetic areas resulting from peripheral nerve lesions. The nerve-fibres in such hyperæsthetic areas should, according to the view here presented, be for the most part of the unmyelinated variety. We propose to investigate both of these questions.

It is interesting to note that throughout his four papers dealing with this question Head conveys the impression that he regards the protopathic group of sensations as primitive in character and the first to appear in phylogenetic development. Now it is well known that nerve-fibres in their earliest phylogenesis are unmyelinated. If our conception is correct a very large part of the afferent nerve-fibres of mammals remain in this primitive relatively undifferentiated state and mediate a relatively primitive and diffuse form of sensation. Other afferent fibres have undergone further differentiation, acquired a myelin sheath, and carry more highly specialized forms of sharply localized sensation. Insulation of the axon by the myelin sheath may play a part in eliminating the element of diffusion so characteristic of the more primitive sensations.

In conclusion, we wish to state that we do not consider that we have done more than formulate a working hypothesis, and shall be satisfied if the considerations here presented stimulate further discussion and investigation.

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EDITOR.

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THE OPHTHALMOLOGICAL OBSERVATIONS OF HUGHLINGS JACKSON AND THEIR BEARING ON NERVOUS AND OTHER DISEASES.[1]

BY JAMES TAYLOR, M.A., M.D., F.R.C.P.

GENTLEMEN,—Let me first in one word thank you for the great honour you have done me in electing me to what I consider the highest position to which one who has worked at neurology may aspire. It is not easy for anyone to wear with grace and distinction the "giant's robe" which the President of this Section must necessarily assume—for my predecessors have all been eminent, and not a few of them great; but such a robe may serve to hide defects and deformities, and at least I shall hope to do nothing unworthy of it, and to hand it over intact and untarnished to a more worthy wearer.

I do not think it can be easy for anyone who was not brought intimately in contact with him to understand the feelings of reverent affection which all of the younger generation who had enjoyed that good fortune always entertained for the great personality of Hughlings Jackson. It may seem presumptuous in me to choose such a subject for my address as that which has been announced, but I should urge that I am not about to criticize, and what I have to say will be more in the nature of an attempt to appreciate some of his work. I trust that what I shall say may have the effect of stimulating interest in his writings so that their wonderful wealth of clinical observation, their accurate scientific method, and their broad philosophical grasp may be better and more generally recognized than they have been as yet.

Jackson's writings are best considered in groups. One of his earliest and most lasting interests was undoubtedly in connexion with ophthalmological and ocular conditions. These ophthalmological interests were, of course, intimately bound up with his interest in the symptomatology of intracranial tumours, and most of his early writing has reference to these subjects. Speech and the alteration of that faculty by disease naturally appealed to his philosophical mind, and I feel sure that all neurologists feel indebted to Dr. Henry Head for the work he has done in placing Jackson's position in reference to this important subject clearly before neurologists. Another division of Jackson's work had reference to paralysis, hemiplegia, chorea and the syphilitic diseases of the nervous system. Still another—intimately associated with the last—dealt with epilepsy in its varied phases, another with vertigo and associated ear conditions. His work on the evolution and dissolution of the nervous system was really a series of philosophical treatises, and his suggestions with reference to the study of disease of the nervous system were severely scientific and yet intensely practical. Among the most interesting of his papers are those designated "Neurological Fragments," and they are, as their name implies, fragments of what one might call intensive neurological investigation and teaching, dealing with an exceedingly varied class of subjects. I need hardly say that this division of subjects is not Jackson's. It is one for which I am entirely responsible, and, as you can understand, one division may overlap another, and many papers find a natural place in more than one of the divisions. But it is convenient to divide his writings in this way so long as we regard the divisions merely as a convenient series of pigeon-holes, and to-night I propose to consider that division which has reference to ophthalmological subjects. This, as will be evident from what I have said, must naturally lead me to refer occasionally to writings which more particularly deal with the symptomatology of intracranial tumours.

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[1] Presidential address delivered at the opening meeting of the Neurological Section of the Royal Society of Medicine, October 28, 1915.

The invention of the ophthalmoscope marks an epoch in medicine. It is nearly always associated with the great name of Helmholtz, but it should be remembered that four years before Helmholtz described his instrument, the ophthalmoscope had really been invented by Babbage, and was only laid aside because an ophthalmic surgeon to whom he showed it failed to discern its possible usefulness, and gave him no encouragement. It is interesting to know that the modification of Helmholtz's instrument soon approximated to the form originally invented by Babbage, and it is this form of instrument, with, of course, modifications and improvements, which is now in general use. Dr. Jackson's friendship and early association with Mr. Jonathan Hutchinson—they were both Yorkshire men, both apprenticed in York, and both held resident posts, not simultaneously, in that city—was no doubt the cause of his becoming early acquainted with Moorfields Eye Hospital, where he worked as Mr. Hutchinson's clinical assistant for several years, and his work here gave an extraordinary stimulus to his interest in ocular and ophthalmological conditions. He has put it on record that he considered it the luckiest thing in his medical life that he began the scientific study of his profession at an ophthalmic hospital [2]. Much of his earliest writings—the result of observations while at Moorfields—are published in the "Moorfields' Hospital Reports," and are mostly concerned with ophthalmoscopic observations. But these observations had almost always reference to the association of ophthalmoscopic changes with medical disease—especially disease of the nervous system—and he was never tired of insisting upon the necessity of routine ophthalmoscopic examination in all such disease.

In an early paper he [21] says: "My object in writing this article is simply that I may contribute from my own field of clinical work a few observations on a class of symptoms, the meaning of which it is most desirable to make out for the sake of advancing our knowledge of diseases of the nervous system. This renders it necessary that I should address myself both to the ophthalmologist and to the general physician, and thus I run a double risk of criticism. If in the slightest degree I do something towards bridging over the special and perhaps somewhat isolated department of ophthalmic medicine with the more general one, I shall have done all I wish to do." The paper to which this is a preface is devoted to a study of defects of sight in brain disease. In this attention is directed to several interesting points. The occurrence of the two kinds of atrophy, one in which the nerve gradually whitens, the other in which it becomes white after certain acute changes, is mentioned, causing what was then called "amaurosis." The difficulty is mentioned of following up and carefully observing such cases because of their tendency to go from one hospital to another, according to the nature of the symptoms. The sudden failure of sight which occurs in some cases of cerebral disease is referred to. Incidentally, he alludes to a danger with which most of us are probably familiar, viz., that an ophthalmological examination, or even treatment, is sometimes blamed for loss of sight. Thus one patient ascribed his loss of sight to a blister which had been applied; another to the use of galvanism; and a third became blind soon after an ophthalmoscopic examination, and ascribed the loss of vision to the examination. In the same paper attention is directed to the headache and vomiting which occur with intracranial tumour, and the danger of mistaking such symptoms for those of gastric disease. Further, the importance and usefulness of ophthalmoscopic examination in cases of hemiplegia, especially for evidence of Bright's disease, and in cases brought in comatose, are insisted upon.

The interest of these early papers of Dr. Jackson's is of two kinds. They embody records of observations of phenomena which were then novel, and the significance of these is set out with a clearness and completeness which left little for subsequent observers to do. But they also contain reflections and remarks which make one feel that the work as a whole is pervaded by a philosophical spirit which breathes life into the dead bones of mere clinical observations. Thus at the beginning of one of the sections of this paper he apologizes for speaking too much of some symptoms which are out of the scope of a journal of ophthalmology [23]: "Much of the interest, however," he says, "depends upon the number of symptoms which can be put together in an intelligent order. Amaurosis may be studied at once with too much intensity and too little breadth. It is to the ophthalmologist a disease of so great importance, calling for particular action on his part, that he may underrate its significance as a symptom in general conditions of the system. But it is when it occurs with other phenomena that we are most likely to discover what optic neuritis means." He then goes on to say that his object in writing is to urge physicians and ophthalmic surgeons to do more work together. Thus if amaurosis and defect of speech are found in the same patient, both the symptoms must be studied by one person. The ophthalmic surgeon can scarcely confine his thoughts to a single sense apparatus, however much his practice may be limited to the defects of that one. To the physician defect of smell, although it may be less important, is of equal significance with defect of sight. Hemiplegia with its allies is the extra-ophthalmological symptom which chiefly deserves the consideration of ophthalmologists because of the frequent ocular or visual anomalies associated with it.

"Odd as it may at first sight seem," he goes on to say, "I feel more and more convinced that there are important reasons why ophthalmologists and those physicians practising in insanity should do much work in common. Not because defects of sight are particularly associated with mental symptoms, nor because insane people may be liable to defects of sight, but because the special study of the evolution of movement (and sensation) is, I think, best begun at ophthalmic hospitals. And so far as we can know anything definite of mind, it is, I suppose, made up of sensory and motor phenomena, the functions of a series of anatomical possibilities in the cerebrum in correspondence with its wide environment, as well as in the spinal cord with its narrow environment. . . . Now nowhere could we better begin to study intelligent movements of muscles than at ophthalmic hospitals. . . . The study of the higher movements, as for words, will, I feel sure, derive great help from the laws of muscular motion which ophthalmologists have been or will be able to establish in their own department of medical practice."

He then outlines a method of medical work: "Were I free," he says, "to work in this metropolis at the physiology of movement, from gross muscular movements towards thought, in the way I should prefer —supposing, to begin with, an ordinary general medical and scientific knowledge—I should study first in the wards and in the out-patient room of a general hospital such symptoms as hemiplegia, chorea, and epilepsy; next, at an ophthalmic hospital, paralysis of the cranial nerves and defects of sight; next at a special hospital for such symptoms as those of locomotor ataxy and certain epileptiform seizures; then at an orthopædic hospital for infantile paralysis and for so-called contractures. Now I would study specially deviations from healthy movements of the eyeballs, and the rudimentary delusional effects they cause. I would, after this work, return to the general hospital, to study defects of speech, delirium, apoplexy, coma, &c. Varieties of defects of speech I should compare and contrast, from difficulty of articulation to incoherence. At this juncture a careful study of laryngeal and throat disease would be most desirable.

"Of course, if anyone were to work at different sections of medical practice, so as merely to add isolated series of facts to one another, he would really make little progress in cultivating his own mind. Such a man would dwell with exaggeration—hurtful to his own organization of medical knowledge—on amaurosis as a defect of sight, and too little on it as a defect of a highly specialized part of universal sensation. He would not improbably neglect altogether such symptoms as defects of smell, which defects, however, are really quite as significant incidents in the revelations which disease makes of function as defects of sight are. Such a student would be altogether unprepared for the last and the very highest medical study, viz., of insanity. Special work at diseases of the mind should, I feel convinced, be begun only after a large real experience of all the special phenomena of motion and sensation that damage to any parts of the nervous system does or may give rise to. I say a real experience, as I suppose a collection of numberless facts, however accurately gathered, is not held to be of itself a real experience. Unless a man can put the particular phenomena he himself sees under more general laws, or unless he tries to do this, he can scarcely be said to know or to be studying a thing in any very valuable sense. The knowledge the ophthalmologist has of muscular disorders of the eye, the knowledge the physician has of defects of articulation, of chorea, and of epilepsy, and that the psychologist has of incoherence and delusions, should aim to be physiological units, each different, but each related to a wide common knowledge of such laws—present or in progress—as those of the evolution of sensation and movement in organisms."

In 1863 Wordsworth [5] had drawn attention to one particular form of amaurosis occurring in tobacco smokers, and Jackson [14] took the opportunity in considering this of raising the question of the rôle of sex in disease, especially in disease of the nervous system, and of offering suggestions for its investigation. Wordsworth had noted the much greater frequency of optic atrophy in men than in women. Hutchinson from a large mass of material had made the same observation. This, as Jackson says, while pointing to some greater liability in the male to optic atrophy, it would be unreasonable to ascribe to tobacco-smoking until more positive and negative evidence had been obtained. He suggested, however, widening the inquiry as to the influence of sex in disease by considering two other diseases—progressive locomotor ataxy and general paralysis of the insane—both of which are diseases of the nervous system rarely found in women [15].

"The widening of the subject," he said, "need not confuse the inquiry by making it more complex; for on a scale sufficiently broad to avoid error, especially the error of attaching too much importance to accidental circumstances, amaurosis can only be studied by an ophthalmologist, progressive locomotor ataxy by those practising generally, and general paralysis by physicians to large asylums. Although, then, there would be more work there would be more workers; and yet when working singly the workers would have a bond in unity of purpose, as their ultimate object would be the same. For although the diseases of the nervous system to be investigated are widely different as groups of physiological symptoms, the pathological question—or at least, the most important part of it—is as to the effect of tobacco-smoking and other sexual habits, peculiarities, or vices, in leading directly or indirectly to degeneration of the elements of a certain tissue—the nervous."

I mention this paper and its suggestions as indicating how widely awake Jackson's mind was to the bearing of recognized ophthalmological conditions on the etiology of nervous disease generally.

Jackson had written as early as 1863 [22] on the existence of optic neuritis without obvious defect of sight, and had frequently repeated this observation. Hutchinson [16] had drawn attention, in 1867, to Jackson's observations, for apparently they had been doubted or even ignored, and Jackson [17] writes that Clifford Allbutt's experience confirms his observations. He goes on to say with quite characteristic modesty: "So far as the observer is concerned the discovery is a very small matter. Any physician who will use the ophthalmoscope by routine must quickly discover the fact. Yet the obvious deduction is really a very important one in practice. This is embodied in the following quotation—italicized in the original—from a paper which I published in 1863 [22]. '*It is, I submit, imperative, in all cases of severe cerebral disease, at all events in cases of an acute kind, to examine the eyes with the ophthalmoscope, whether the patient complains of defect of sight or not.*"

"I should now make the statement much stronger by adding, '*even if he affirms that he can see well, and if he read small type readily.*'

"I have occasionally had the somewhat painful feeling that the accuracy of my ophthalmoscopic examination has been doubted by physicians whose opinion I highly value, when I have declared that patients with severe cerebral disease, who seemed to see quite well, had inflamed optic disks." This point Jackson had to insist upon many times before it was accepted, and we are apt to forget that what to us is now commonplace required such reiteration and insistence.

A question which was frequently discussed at this time was, Does disease of the cerebellum lead to loss of sight? Brown-Séquard had stated that it was certainly often associated with this, and he expressed the opinion that it was the result of reflex action from some irritant. Jackson [1] discusses the question, and clearly expresses the opinion that disease of the cerebellum, *per se*, does not produce blindness, any more than disease of the cerebrum, but that tumour in either region may and often does, by setting up optic neuritis—"the result of a local encephalitis." "Whilst destruction of no part of the cerebrum or cerebellum leads to loss, not even to defect of sight, irritation by tumour in any part of either may lead to inflammation of the optic nerves, on which inflammation, blindness usually—not always—follows."

At this stage, in 1871 [18], Jackson published a lecture on optic neuritis in intracranial disease, in which he sums up in a succinct form much of what he had been teaching for several years. He points out the value of the ophthalmoscope in diagnosis: that morbid ophthalmoscopic signs are rarely absent in amaurosis from cerebral disease, that optic neuritis is the commonest ophthalmological condition in cases of cerebral disease, and is as important in the diagnosis of cerebral disease as is hemiplegia. He then describes the stages of optic neuritis—the onset, the second stage with great swelling, the third in which atrophy is commencing, and the fourth that of permanent atrophy. He also again, what he earlier insisted upon, points out that in some cases of optic neuritis there is no defect of sight, that optic neuritis is almost invariably double, and that paroxysmal failure of sight occurs in cases of optic neuritis. Occasionally, also, it is pointed out, there is rapid and permanent failure of sight. With reference to the nature of the intracranial disease most often associated with double optic neuritis he makes two statements:—

(1) There is usually a gross change, or "coarse" disease, a lump of something, an adventitious product.

(2) Double optic neuritis does not point to any particular *kind* of coarse disease, but simply to coarse disease of *some* kind. There are certain conditions under which double optic neuritis scarcely ever occurs, e.g., chronic and general convulsive attacks, a condition of hemiplegia from local softening, &c., and he points out that optic neuritis is not a localizing sign, and then discusses the different theories as to the manner in which an adventitious product in the cerebrum leads to optic neuritis. He concludes by begging his readers to remember three things: (1) That optic neuritis frequently exists when the patient can read the smallest type; (2) that the ophthalmoscopic appearances vary extremely in degree in cases of adventitious products within the cranium, and that the appearances vary much at different stages in the same case; and (3) that one should never omit to use the ophthalmoscope when a patient has severe and continued headache.

During the next few years Jackson continued from time to time his ophthalmological contributions. His "Physician's Notes on Ophthalmology" [25] are a storehouse of interesting records of many cases carefully and systematically observed, and of even more valuable suggestions. He also published anomalous cases [26], one of large cerebral tumour *without* optic neuritis, another [27] of double optic neuritis without cerebral tumour, and he also recorded [32] a case of recovery from double optic neuritis. It is clear, as will be seen, that he recognized the curious variability in the incidence of ophthalmological signs of severe cerebral disease; and that (1) intracranial tumour may be present without optic neuritis; (2) optic neuritis may be present and yet no tumour be found; and (3) optic neuritis does not necessarily result in blindness, although there is usually great danger of its leading to impairment of sight, and yet that occasionally it passes off without causing any impairment of vision.

The Annual Oration to the Medical Society of London [2], which he delivered in 1877, is perhaps one of the most characteristic and most finished of Jackson's addresses. It is an interesting combination of that scientific knowledge, clinical observation and philosophical suggestion to which reference has already been made. It sums up in a very clear way much of what Jackson had been engaged in teaching for many years, and more especially it dwells upon the work of the ophthalmic surgeon as a training in discipline and surgical skill, and as affording opportunity for observation of ocular palsies and their effects, of the anomalies of refraction, of the actual state of a nerve and its termination, and of the condition of the blood-vessels and the retina generally. He emphasizes the importance of recognizing the effects which refractive errors may produce. Thus astigmatism may produce "confusion of vision" because of the difference in the distinctness in different meridians; hypermetropia may cause, and has actually caused, symptoms simulating those of brain disease, such as headache and squinting, and observation of hypermetropic disks is of the utmost importance because of the close resemblance they bear to the inflamed disk associated with intracranial tumour. The phenomena of what was then called diphtherial amaurosis as they are revealed to the ophthalmic surgeon are also dealt with, and their relationship to affection of the ganglia is pointed out, and the interesting question is raised whether in diphtheria there is, as affecting hearing, something analogous to paralysis of accommodation in the eye.

The study of paralysis of the ocular muscles is considered. This naturally leads to a consideration of vertigo generally and also of defects of co-ordination. The vertigo which occurs from paralysis of an ocular muscle is not, it is pointed out, the result of diplopia, but of erroneous projection. A patient suffering from paralysis of the right external rectus, if the left eye is covered and he is asked to touch an object placed to the right of the middle line, will miss the object, his hand going to the right of it. That is, the patient judges by the activity of the centre for movement—"the hand is *misinformed*," to speak metaphorically, or to use an incoherent simile, "makes a false step." It is not difficult to see the analogy between such an action and actual reeling. During walking there will be many actual false steps due to the misleading information which the centre for the impossible movement of the eye supplies. It is inaccurate to say that vertigo *causes* disorder of co-ordination: it is itself disorder of co-ordination. The only difference between vertigo *without* manifestation and vertigo *with* the outward manifestation of a "reel" is one of degree. In the former there are slight central changes and in the other strong central changes, leading to peripheral effects. There is thus an analogy between those two conditions, or rather degrees, of vertigo and the two conditions of speech—speaking internally and speaking articulately.

He then proceeds to draw a distinction between sensation and the physical process occurring along with it. "The use of the word sensation both for a state of consciousness and for a state of the nervous centres produces the same kind of confusion as the use of the expression that the sun rises in the east would do in an exposition of the movements of the solar system!"

"The facts of ocular vertigo, with other allied facts from other departments of medical practice, throw light on psychico-physical operations. Speaking generally, they help to show that ideas arise, not only during energizing of sensory centres, which everybody admits, but during energizing of motor centres, which scarcely anybody seems inclined to believe. We have seen that the patient judges of the positions of objects by central discharges which do not actually displace his retinal images. Now the extension of an object is really made up of relations of innumerable positions. To give a particular illustration, the facts of ocular vertigo are of inestimable value in supporting the doctrine that the anatomical substratum of a word is a motor, an articulatory process."

Another aspect of the matter is next considered—viz., that in some cases of paralysis of ocular muscles the size of the object is altered. The fact is referred to that if we impress the retina with a scarlet object the after-image will be altered in size with accommodative efforts. There is in this phenomenon, of course, a motor change only, for the retinal area affected is unaltered. The shape of objects also, it is pointed out, can be altered in some degree. Thus if we obtain the after-image of a circle and project this on to an inclined sheet of paper, the spectral circle becomes oval. The case is also mentioned of a medical man subject to migraine, who observed that he could alter by accommodative efforts the size of the visual spectra which preceded his headache. And in some cases of epilepsy there is a warning in the alteration of the size of objects: mostly they get bigger and nearer—"the walls of the room seemed to come nearer," as one patient expressed it. All these cases, except probably that of epilepsy, it is claimed, show that estimation of the size of objects depends upon activity of a motor element only. And the fact that in paralysis of the ciliary muscle by atropine or by disease objects appear smaller shows that activity of motor centres may cause alteration in the size of an object when actual movement is impossible.

"These facts," he goes on to say, "like those given in speaking of vertigo, show plainly, I think, the inseparable connexion of motor activity with sensory activity in ideation. They show that, whilst the colour—the secondary or dynamical quality of an object—is a sensory affair, its size and shape, its primary or statical quality, is a motor affair. If so, then ophthalmological facts are of inestimable value, not only as bearing on an important psychological problem, but as bearing on the most important of all questions whatever in psychology; they demonstrate that the estimation of the extension of objects is due to motor activity; and they show that activity of motor centres will suffice; and thus they lend support to the doctrine that, in remembering the shape of an object, there is slight activity of central motor centres just as much as there is slight activity of sensory centres in remembering its colour."

Similarly in reference to locomotor ataxy he argues against the hypothesis of a "special co-ordinating centre." He explains the ataxy by supposing that the current developed does not—on account of diseased tracts—react with full effect on the muscle most concerned with accurate locomotion, the peroneus longus, but overflows into the tibialis anticus and so produces in it over-action—really the same result as what is called "secondary deviation" in certain conditions of ocular paralysis. There is, in short, over-estimation of the intended, but underdone, peroneal movement and actual overdoing of the tibialis anticus movement. An analogous condition had been pointed out by Duchenne in the case of a hand wasted in regard to its flexors. When such flexors are atrophied, and a strong attempt is made to close the fingers, the actual effect is over-extension of the fingers, the current intended for the paralysed flexors actually overflowing into the extensors.

With reference to sensory affection of the eyes, it is pointed out that hemiopia is a sensory symptom which is the strict analogue of the motor symptom, lateral deviation of the eyes, and it is frequently associated with hemianæsthesia. Sight sensations, it is pointed out, are more frequently precursors of epileptic attacks than any other special sense warnings except touch. Smell is next, but auditory warning is rare. That sight should be the most frequent is what one would expect, for most ideation is carried on in visual ideas. A patient may have sight sensations, e.g., coloured vision, long before he has epileptic attacks ushered in by such sight sensations.

He then proceeds to refer to the use of the ophthalmoscope, pointing out its importance, for in cases of defective vision associated with intracranial disease there is nearly always change visible with the ophthalmoscope. Yet it is very important not to make too much of slight changes. Imperfect observation may detect "congestion" and "anæmia" of the disk when physiological redness and pallor are the true conditions. "Some observers," he says, "are really inferring when they should be only looking." He also pointed out that those who do not use the ophthalmoscope are apt to miss important changes and perhaps to misinterpret symptoms. It must be remembered that it is exceedingly common for a patient with intense optic neuritis to see quite well, and if such a patient died and a cerebral tumour were found, unless an ophthalmoscopic examination had been made the presence of inflamed disks would have been undiscovered, and the significance of such a condition missed. An illustration of the converse of this was the case of a patient who became almost blind and died. Post mortem, a condition of disease of the optic thalamus was discovered. In such a case the blindness might have been ascribed to the disease in the optic thalamus—quite erroneously, had it not been discovered by the ophthalmoscope that in each retina there were the extensive changes of albuminuric retinitis.

Optic atrophy also, it is pointed out, is important in reference to examination with the ophthalmoscope. It often occurs in tabes dorsalis and in general paralysis of the insane. It may occur with or without pupil changes, with or without pain, with or without ataxy. And, of course, it is not a necessary sign in tabes dorsalis.

Another condition noted with the ophthalmoscope is retinal embolism. This may be associated with evidence of cerebral embolism, and observation with the ophthalmoscope is important, as otherwise the optic atrophy, the result of embolism of the central artery of the retina, might be mistakenly supposed to be the result of cerebral disease.

Similarly also, it is pointed out, the ophthalmoscope may render visible other tissue changes in the fundus, such as those resulting from tubercle and syphilis and from Bright's disease. But it has to be noted that the ophthalmoscopic appearances of Bright's disease are often closely simulated by those occurring in intracranial tumour, while conditions really depending upon Bright's disease are often such as strongly to suggest the condition of the fundus occurring with intracranial tumour. Reference also was made to the visible arterial pulse in the fundus found in association with aortic regurgitation and to the retinal hæmorrhages occurring in some cases of purpura.

In the paper [13] "Remarks on the Routine Use of the Ophthalmoscope in Cerebral Disease" he returns to a subject on which he had written early and often. He makes out a strong argument in favour of this procedure, and it may be said that it is chiefly due to his insistent teaching that the ophthalmoscope is now in such constant use in the hands of neurologists.

He quotes physicians and ophthalmic surgeons in support of his repeated allegation that marked optic neuritis may be present with perfect vision. Numerous cases are cited in which marked optic neuritis was present, yet no change was discoverable either in the acuity of vision or the extent of the fields. Yet in many of these cases post-mortem examination revealed the presence of intracranial tumour or the subsequent progress resulted in blindness. The practical importance of the discovery of optic neuritis is pointed out not only with reference to prognosis but also in regard to treatment, and it is again pointed out that neuritis may coexist with good vision and may pass off, leaving sight good.

Jackson published an important paper [28] on eye symptoms and tabes. It may be said that nearly all he tells us on the subject is now commonplace knowledge, but at the time it was written the paper set out points not then clearly grasped. He mentions three non-ocular signs in tabes—lightning pains, loss of knee-jerks, and ataxic gait—and three ocular—paralysis of oculo-motor nerves, pupil changes, especially the Argyll-Robertson phenomenon, and optic atrophy—whilst he clearly indicated that not all of these occurred simultaneously in many cases of tabes, but that two or three of them not infrequently occurred in the same case. But the important point was the recognition of any one of them in a patient as indicating the likely nature of the case and as suggesting the possible presence of others if carefully looked for.

In the same volume [28] there is the record of a very interesting discussion on "optic neuritis in intracranial disease" in which Dr. Jackson took part. His contribution is full of facts, and has many suggestive questions as well as statements. It is in six sections. The first deals with optic neuritis ophthalmoscopically. He says he used arbitrarily to make two stages in optic neuritis: (1) slight œdematous swelling, and (2) a climax of extensive great swelling, with hæmorrhages and strangulation of vessels, but he avers his belief that there is but one kind of optic neuritis from intracranial disease. But he points out again the simulation of the optic neuritis of intracranial disease by the condition found in Bright's disease, and mentions a case in which he himself had made the diagnosis of brain disease simply from the appearance of the fundus oculi, yet post mortem no such disease was found, but the patient, a young boy, was found to have granular kidneys. He asks, Can the fundus condition in such cases, as well as in cases of meningitis, be definitely distinguished from that in cases of intracranial tumour?

The next section (2) deals with clinical facts. Uniocular optic neuritis rarely occurs in cases of intracranial tumour. When it does, what is its significance? Does it ever result from cerebellar disease? He says he believes the diagnostic value of optic neuritis is not different whether sight be good, defective or bad. But physicians see more cases in which the vision is good than with even defect of sight. He also refers to the occasional temporary failure of sight and the rapid permanent failure of sight, which occur in some cases, and inquires as to their significance.

The third section is devoted to a consideration of optic neuritis with other symptoms, non-localizing, such as vomiting, headache, &c., and localizing, such as unilateral convulsions, slowly progressive unilateral weakness of limbs, head enlargement, sudden hemiplegia, as from hæmorrhage into a tumour, reeling gait in cerebellar cases, and cranial nerve palsies, and he points out that optic neuritis does not occur in ordinary epilepsy, with complete aphasia, or with extreme mental conditions.

Section 4 deals with the diagnostic and non-diagnostic value of optic neuritis. It usually occurs with intracranial tumour or some other adventitious product. The mass may be in any part of the encephalon, although probably tumour in the medulla oblongata rarely produces optic neuritis. Yet it may be absent with tumour in almost any part, i.e., "optic neuritis occurs with tumour in almost any part of the encephalon, and may not occur with tumour in many parts of it." Also it may occur late in cases of cerebral tumour, or it may appear late and pass off, the patient dying ultimately of tumour. It is rarely found with softening from clot, it is not caused by mere destruction of any part of the encephalon, and, as has been insisted on before, it may be unattended with impairment of sight. Optic neuritis points to the general nature of the local disease, not to its particular nature. It means, as a rule, "foreign body," and, so far as the optic neuritis is concerned, that produced by a syphilitic or gliomatous "lump" is not to be distinguished from that produced by any other kind of foreign body.

It is important to distinguish between the development of neuritis from a foreign body and loss of sight from a destructive lesion. Thus hemiopia is usually the result of a destructive lesion and in itself implies no change in the fundus. Of course, a tumour so situated as to cause hemiopia will probably cause also optic neuritis. Yet in the ordinary hemiopia from a hæmorrhage or clot, fundal changes do not occur. "The several different lines of evidence agree in converging to the conclusion that optic neuritis in cases of intracranial adventitious products is to be looked upon as resulting secondarily from such products in their general, or more correctly in their abstract, character—that of 'foreign bodies.'"

Section 5 deals with the various hypotheses as to the mode of production of optic neuritis.

(1) von Graefe's idea that it is produced by raised intracranial pressure inducing venous congestion in the central vessels of the optic nerve. Against this is the fact that a small tumour may cause intense optic neuritis and that a large hæmorrhage may not cause, does not cause, optic neuritis.

(2) Hypothesis of Schmidt, which ascribes optic neuritis to distension of the optic nerve sheaths.

(3) Hypothesis of Benedict, first hinted at by Schneller, viz., that of reflex vasomotor action (analogy of convulsion) by induced instability in grey matter, and action in influencing vessels in optic nerves. Loss of sight, temporary and sudden, does occur with optic neuritis, and often occurs also at onset of a convulsion. Secondary changes in the brain are certainly produced by tumour as is shown by epileptiform seizures. Optic neuritis is thus supposed to be a doubly indirect result of tumour. The sequence is supposed to be: tumour, changes of instability, effects produced by the latter on muscular-walled arteries

of optic nerves and centres. Jackson considers this hypothesis, the vasomotor one, *the most plausible.*

The sixth section deals with treatment. Optic neuritis, although, secondary to tumour, becomes independent of tumour, becomes autonomous. He supposes that if we were able to reverse all central changes which had led to optic neuritis the optic neuritis would remain for treatment. He thinks it clear that the neuritis does become autonomous because atrophy which is permanent ensues when all other signs of cerebral disease are gone, when this disease has become quiescent. The best time for treatment is early. Hence the importance of early recognition by the use of the ophthalmoscope.

Jackson reported a case [29] of ocular movements produced by pressure on a diseased ear. He had at various times written on Ménière's disease and on the vertigo occurring in that condition. Vertigo, indeed, was a symptom which always excited his interest, whether it was ocular or auditory in origin, or if it were associated with epilepsy.

The patient was a woman with old-standing otorrhœa, in whom pressure on the right tragus produced movements of eyes, slow, to the *left*, followed by quicker movement back to the *right.* Objects appeared to move to *left*, i.e., with slower jerks. When she improved and jerks to *right* became slower, she was able to see objects move also to right.

He supposes that pressure on the tragus was transmitted by the chain of bones to the contents of the semicircular canals, and changes in the canals were propagated through cerebrum or cerebellum—possibly both—to ocular muscles. The case is also important as showing dependence of vertigo on ear disease. She was subject to giddiness and even occasional reeling.

His Bowman Lecture was delivered before the Ophthalmological Society on November 13, 1885. It is a philosophical treatise on Spencerian lines, but with definite practical application. Differentiation or division of labour, he says, is a universal law, and the body medical is, of course, subject to this law. Specialism is therefore a natural outcome, but specialists have to justify their differentiation. But the modern doctrine of evolution involves more than differentiation. The other factors are increasing definiteness, increasing integration, and increasing co-operation. The ophthalmic surgeon has justified himself in the factor of definiteness by his work on ocular muscles and errors of refraction; he has also justified himself in the factor of integration. Thus Argyll-Robertson in the pupil symptom has given to neurologists not merely a new symptom but a means of investigation of several important diseases; and neurologists like Westphal and Erb have paid back the debt by showing the diagnostic value of the knee-jerk. The necessity of the fourth factor—co-operation—is shown, e.g., in such a case as one of hypermetropic headaches. The neurologist may be consulted for the headache, and he ought to be able to suspect hypermetropia as a cause. But only the skilled ophthalmic surgeon can estimate it precisely and correct it accurately.

The complexity of some nervous diseases is, he says, often underrated. Epileptic paroxysms and their after-conditions are an illustration of such complexity. When its symptomatology is considered, there is shown the necessity of different, definite, wide knowledge and co-operative work. Ophthalmic surgeons, aural surgeons, alienists and psychologists can all contribute to our knowledge of the symptomatology of the disease. Thus the ophthalmic surgeon may investigate the fundus. He may study visual auræ—colour warnings or "seeing faces"; the condition of the pupil will be observed. Thus in uræmic convulsions pupils may be dilated and become smaller after the paroxysm. Vertigo also will have to be investigated by the ophthalmic surgeon as well as the aural. The condition of the eyes after a paroxysm also will be observed. Beevor found they were deviated to the side opposite to that to which they deviated at the commencement of an attack. Another curious eye symptom in the epileptic aura will demand attention, viz., the apparent alteration in the size and distance of objects, comparable with the micropsia after the instillation of atropine. Such are some of the phenomena requiring investigation in epilepsy at the hands of an ophthalmic surgeon.

But there are other non-ophthalmological symptoms of epileptic paroxysms, especially of those of organic parts. These are an important part of the evidence, showing that the "organ of mind" (the highest centres) represents all parts of the body. The highest centres are the organs of will, memory, reason and emotion, the four elements of mind, i.e., of consciousness. "No wonder the epileptic discharge, beginning in some part of the 'organ of mind,' produces universal bodily effects, animal and organic. Unless retinal impressions corresponding to colour of objects, and ocular movements corresponding to shape of objects, be represented in the highest centres, how are we to account for the physical basis of visual perception and ideation? If the organic parts are not represented in the highest centres, an emotional manifestation, say of fear, is unaccountable, and so, too, are the physical symptoms of some cases of melancholia, the insanity of fear (anxiety, depression, &c., being only 'fear spread out thin.')"

Much attention is given to the movements of chewing or tasting, swallowing, &c., in association with slight fits, and especially associated with the "dreamy state." He expresses the belief that they, as well as another phenomenon occurring in epileptic warning—the apparent alteration in size or distance of external objects—are the indirect (reflex) results of epileptic discharges of sensory elements. He also believes the "dreamy state," "seeing faces," "hearing voices," and such elaborate mental states arise during slightly raised discharges of healthy nervous arrangements, i.e., arrangements untouched by the epileptic discharge. The "dreamy state" he regards as a very voluminous mental state—not an aura, although it has been called an "intellectual aura"—and he believes that the subjective sensations—smell, taste (chewing movements, &c.), and the epigastric sensations—most often occur in cases of epilepsy in which there is a dreamy state, and depend upon lesions in the cortical area supplied by the posterior cerebral artery. When the warnings are of colours or sounds, the lesion is probably in the cortical area of the middle cerebral, and he enunciates his belief that the nervous changes (discharging lesion) in epilepsy are not primarily but only secondarily nervous; that in most cases they are secondary to embolism or thrombosis of small arterial branches.

Attention is next directed to the after-condition of epileptic paroxysms. Insanity may occur in at least two forms—post-epileptic "unconsciousness" with mania and acute dementia (coma). Physically this is universal paralysis, and paralytic phenomena are present, such as transitory lateral deviation of the eyes, exaggerated knee-jerks, and ankle clonus.

For the study of such a complex disease, therefore, the neurologist must avail himself of the work done by different specialists. "We require many different kinds of *definite* or technical knowledge. All the sagacity in the world will not suffice either for practical ends or for the scientific investigations of complex problems without technical knowledge."

The only method of investigating such a complex disease is that by the use of hypothesis, and it is suggested that the hypothesis of evolution should be adopted for this purpose. According to this all parts of the body are represented in each of three levels of evolution.

"Representation increases in differentiation, definiteness, integration and co-operation from lowest centres to highest centres. The 'organ of mind' is nothing else than a series of centres representing, or what is the same thing co-ordinating, all parts of the body 'from nose to feet' in greatest complexity. The evolutionist does not attempt the marvellous feat of 'getting the mind out of the body,'" he only tries to get the physical basis of mind (highest centres) out of the rest of the body.

To test this evolutionary hypothesis one should use the experiments which disease makes in effecting dissolution. Thus paralysis of the ocular muscles from affection of their nerve trunks is an example of disease of the lowest level. So are ophthalmoplegia externa and interna. Lateral deviation of the eyes occurs in lesions of the middle motor level, and deviation of the eyes in and after epileptic fits is probably the result of lesions in the physical bases of innumerable visual ideas and other mental states.

It is also suggested that we may have atrophies of cells to organic parts analogous to atrophies of anterior horn-cells. Thus diabetes may be the result of a nuclear atrophy of cells of that part of the great vasomotor centre which especially governs the hepatic artery. Similarly Graves's disease and myxœdema may be of similar etiology. These, of course, we regard as being lesions of the lowest evolutionary level. But these examples must be compared and contrasted with the symptoms of negative lesions of the highest level. After an epileptic fit the condition is one of nearly universal paralysis (dissolution effected).

"Since the epileptic discharge starting in the highest level produces effects in nearly all peripheral parts, currents from this primary discharge must have traversed and discharged more or less of the middle and lowest centres in order to get at the periphery. Hence the paralytic condition after a severe fit wil be a very compound one, all orders of centres being somewhat affected, but in different degrees."

In conclusion, it is urged that the neurologist, by availing himself of the work of ophthalmic, laryngeal, aural, &c., surgeons along the lowest line of evolution, by working himself at the middle, by co-operating with alienists in the study of diseases of the highest level, may hope to justify his specialism. And the different workers may hope to add different knowledge, to attain to more definite knowledge, to further the integration of general medical knowledge, and to lead to higher and more methodical co-operation of different workers.

In 1886 there was a discussion [30] at the Ophthalmological Society on Graves's disease. Jackson's contribution was brief but important. He said that he attached much importance to von Graefe's sign, although he had seen well-marked cases of Graves's disease in which it did not occur, mentioning in illustration two cases in sisters. He suggested a comparative study of such cases with some other family diseases. He mentioned that in the recent cases of Graves's disease he had seen, eight in number, the right lobe of the thyroid was the larger, a fact bearing on the question of the central pathology, as W. A. Fitzgerald had suggested, for the right vagus had, in some lower animals, more inhibitory influence on the heart than the left. He also expressed the opinion that the hypothesis of a central pathology was the most probable, and referred to the experiments of Brown-Séquard and Filehne on the production of exophthalmos by injuries of the restiform body. In all cases of fatal Graves's disease the medulla oblongata and pons Varolii should be carefully examined microscopically. He referred to a case mentioned by Warner and Bristowe in which there was *ophthalmoplegia externa*, and a case which he had seen complicated with asthmatic paroxysms, and one with paroxysms of right facial spasm. Pavy had noted the association of Graves's disease with diabetes. In the so-called complicated cases the association might be accidental, but they deserved investigation. He did not remember seeing Graves's disease in a man.

In his Presidential address [31] to the Ophthalmological Society, Jackson says that no department of medicine had greater attraction for him than ophthalmology. It was the first subject at which he specially worked after his student life, and he gratefully acknowledges—as he so often does in his writings—his indebtedness to the example and teaching of Jonathan Hutchinson while he was his clinical assistant at Moorfields. He emphasizes the benefits of the opportunities for being well disciplined in exact observation which an ophthalmic hospital affords, and the effect of such discipline in teaching students to avoid vague and indefinite statements.

"Since six cranial nerves and the sympathetic nerve," he goes on to say, "supply the eyeball and its apparatus, it is evident that without a good knowledge of eye diseases the thorough investigation of very many morbid affections of the nervous system is not to be methodically carried out. . . . Unless the physician uses the ophthalmoscope by routine he will often enough overlook the best evidence—and I am convinced in many cases the only decisive evidence—of gross organic disease of the brain there is to be had; and if, as is most often the case in a physician's practice, sight be good, he will not surmise that there is anything wrong with his patient's optic nerves, and will very likely be incredulous when someone who has looked at them tells him that there is swelling of the disk." Further, without special knowledge, paralysis of the superior oblique may be overlooked, and the symptom—vertigo—for which the patient seeks advice may be misinterpreted. So also hypermetropia as a cause of headache may be overlooked and the hypermetropic disk, unless one is familiar with its appearance, may be regarded as a condition of optic neuritis indicating serious brain disease. So that "without a good knowledge of ophthalmology a methodical investigation of diseases of the nervous system is not merely difficult but impossible."

He also suggests that cases of uniocular optic neuritis with tumour of one hemisphere are the cases which will throw most light on the process by which optic neuritis results from disease in various parts of the encephalon. "At present," he says, "there are only hypotheses as to this process," although he regards the hypothesis of vasomotor action as the "most plausible." Various other conditions have to be accounted for: the epileptiform paroxysms, the sudden temporary loss of sight, the vomiting and slowing of the pulse, the tendency to sudden death, and the question is asked, Do these symptoms not dependent upon optic neuritis depend upon a similar morbid condition in other nerves, such as the pneumogastric and its centres, as suggested by Thomas Buzzard?

The integration of ophthalmological knowledge may also help, by the study of nystagmus, to elucidate the tremor of Graves's disease and the various occupation spasms such as writer's cramp, and the work of the ophthalmic surgeon in ocular paralysis may help to the interpretation of inco-ordination produced by destructive lesions of nervous centres in animals.

Further, in the study of eye conditions we are most likely to be able to trace the ascending complexity in the evolution of movement from ocular muscles to their representation in most complex movements in the highest motor centres, i.e., in the physical bases of visual ideas. Such a study would do much towards showing that the organ of mind is sensorimotor, and we should then place the study of epilepsy proper and insanity on a realistic basis. Further, the great importance of a study of ocular paralysis is insisted upon in reference to the question whether mental states do or do not occur with the "outgoing" currents as well as with the "ingoing" current, and this question should interest alienist physicians in ophthalmological work. And physicians and aural surgeons especially can help ophthalmology by showing and investigating cases such as spasmus nutans with nystagmus; tetanus, as to whether ocular muscles are implicated in this disease; and cases of auditory vertigo, especially if associated with ocular movements. He concludes with a quotation from a paper by James Anderson [20]: "It seems to me the best and most hopeful feature of ophthalmology that it has relations, closer, or more remote, with every branch of medicine and surgery, indeed with almost every branch of science."

In the introductory part of this address I referred to the series of "Neurological Fragments" published by Dr. Jackson. The first [6] of those deals with a very interesting condition. Mendel [19], as a result of a series of experiments on animals, formulated the hypothesis that the oculo-facial group of muscles (frontalis, corrugator supercilii and orbicularis palpebrarum) is really innervated from the third nucleus, although the fibres to these muscles are distributed in the seventh. A case published by Tooth and Aldren Turner [3] lent support to this view. In the first neurological fragment Dr. Jackson considers the same point, and gives an account of the investigation of two cases of ocular palsy with ptosis in which weakness of the orbicularis could be demonstrated. He alludes to the fact mentioned by Duchenne that the orbicularis palpebrarum really consists of four muscles, and to the fact mentioned by Fuchs that with regard to closure of the lids we have to distinguish between moderate closure, as in winking and sleeping, and the process of screwing the lids tightly together. No doubt, Jackson says, the muscle is represented in several places in the central nervous system according as it takes a share in different movements. He suggests that one of these places for the representation of the orbicularis palpebrarum is that in which the external ocular movements for directing the eyes for the estimation of distance are represented. Presumably the peering movements of a short-sighted person, movements helping him to see better, will be represented along with certain movements of the eyeballs, and as the third nerve is that chiefly implicated in ocular movements the representation of the orbicularis so far as these movements are concerned will be most probably in the nucleus of the third nerve.

In the second neurological fragment [7] he refers to the interesting class of cases in which with congenital ptosis elevation of the eyelids can be produced by masticatory movements, but especially by lateral movements of the jaws produced by the external pterygoid muscles, a fact suggesting, as was pointed out by a committee which investigated Marcus Gunn's case of this character, that in such cases the levator palpebrarum is supplied both from the nucleus of the third nerve and also from the external pterygoid portions of the nucleus of the fifth nerve. "It seems pretty clear," he says, "that the point of emergence of a nerve trunk from the central nervous system is a very untrustworthy clue to the nuclear origin of the several fibres making it up."

In the third neurological fragment [9] suggestions are made as to the use of cocaine for the investigation of abnormal conditions of the eyes, taking advantage especially of its characteristic, that it stimulates the endings of the sympathetic, dilating the pupil and widening the ocular aperture. He suggests, e.g., observations on the effect of cocaine on the Argyll-Robertson pupil and on the conditions of suddenly occurring immobility, under all conditions, of one pupil, with dilatation and loss of accommodation of the same eye, and on the reaction to cocaine of the pupil at different ages. It should also be used, he suggests, in cases of congenital ptosis, in order to ascertain, by its effects on Müller's muscle (stimulation of which in the normal condition widens the ocular aperture), whether that is affected in those cases as well as the levator palpebrarum.

The fourth neurological fragment [10] deals with the condition of the pupil and eyelids in cases of paralysis of the cervical sympathetic. Long ago [12], he mentions, Jonathan Hutchinson drew attention to the peculiarities of ocular paralysis from lesion of the cervical sympathetic. In cases of fracture dislocation of the cervical spine the pupils may be of equal size and may contract to light and during accommodation, yet if the investigation is not pushed further a very interesting sign may be missed. After speaking of paralysis of the "dilating" fibres of the pupil in such cases, Mr. Hutchinson wrote: "This is a very important and valuable symptom. It occurs only when the injury is in the cervical or upper dorsal region. The pupil is neither dilated nor much contracted. It is simply unable to dilate. Unless carefully examined the myosis, being so slight in degree, may easily be overlooked. The plan is to examine the eyes in shade, and you will then find that the pupils remain just of the same size as they were when exposed to light. Sometimes one pupil is more definitely contracted than the other." He mentions a case of a wound of the neck under the care of the late Mr. Marcus Beck, in which only one eye was thus affected. The pupil of the affected eye did not dilate, as did that of the other eye, when the patient's back was turned to the window, nor did it dilate on faradic excitation of the skin, and Dr. Head found that cocaine had no action on the pupil or on the lids of the faulty eye. Cases of rupture of the brachial plexus, cases of thoracic aneurysm, cases of wounds and tumours of the neck, should be investigated in reference to this point, and so also should cases in which the small muscles of the hand are affected, especially cases of syringomyelia and of anterior poliomyelitis. He had referred in the Bowman Lecture to a case which he supposed to be a case of progressive muscular atrophy in which there was affection of one pupil, believed to betoken paralysis of so-called dilator fibres given off by the cervical sympathetic nerve to the iris and to Müller's muscle, a case which he now, by reference to the original notes, had little doubt was one of syringomyelia. He had been puzzled by finding no such pupil abnormality in undoubted cases of the Aran-Duchenne type of progressive muscular atrophy. He suggests that fibres ultimately passing in the cervical sympathetic to palpebro-ocular structures may not go through anterior horns, and if they do they may not atrophy in Aran-Duchenne progressive muscular atrophy. The effect of cocaine in such cases and in cases of anterior poliomyelitis, the hands being affected, may throw light on the exact course of the fibres in question.

In a paper, the last which he published [11] "On Some Abnormalities of Ocular Movements" written in association with Leslie Paton, a series of very interesting observations and experiments is described, undertaken with the view of elucidating whether "erroneous projection" which occurs in paralysis of, e.g., an external rectus muscle is the result of an increased outgoing current, as held by Bain and Wundt, or of an ingoing current as suggested by Müller, and a little later by James.

He remarks that movements of both eyes are represented in each half of the brain, but the lateral movements of both eyes to one or other side are most specially represented. But as he had remarked [8]: "Every movement called voluntary will be represented in the cerebral hemispheres, and thus there must be representation in them of all ocular movements a man can effect when he tries." "I asked Dr. Risien Russell," he says, "to make researches on the matter. At my suggestion he cut the right external rectus and the left internal rectus of a monkey, thus rendering movements of the two eyes to the right impossible. Upon faradic excitation of the animal's left cortical area, the eyes turned directly upwards or directly downwards according to the particular part of the eye area stimulated" [4]. This suggestion of Jackson's has always seemed to me one of the simplest yet most illuminating which he ever made.

The basis of the experiments carried out by Paton was the artificial simulation of paralysis of an external rectus muscle by an appropriate prism. So far as secondary deviation and erroneous projection are concerned there will be no material difference between prism paralysis and ordinary external rectus paralysis. But it is obvious that as all the nervous tracts, ingoing and outgoing, remain intact, such an experiment can offer no support either to the outgoing or ingoing theory. But an interesting and almost crucial observation was made on a man who had paralysis of his left third nerve, paralysis of the right sixth and paresis of the right third nerve, upward, downward and inward movements being defective. The varied observations on erroneous projection need not be detailed, but the result of these, in the words of the observer, "seemed to be definitely opposed to the hypothesis that the false projection in the case of one eye where one of the muscles is paralysed may be due to impulses coming up from the muscles of the sound eye," for as soon as one of the paralysed muscles of the right eye was called into action the patient got false projection in the direction of action of the respective muscles. Such an effect in this case could not be the result of afferent impulses from the other (the left) eye, which was covered, since the left eye was always in a position of left deviation.

It is not easy to sum up briefly the outcome of Jackson's teaching in the various papers to which reference has been made. From the practical point of view his insistence upon the routine use of the ophthalmoscope is to be noted because of the importance of its revelations in reference to diagnosis, prognosis, and treatment, and because of the scientific bearing of its discoveries on the symptomatology of intracranial and other diseases. Optic neuritis itself, he asserts, has no localizing value, but he says that the study of instances of uniocular optic neuritis and the careful examination by every means in our power of such cases may furnish us with a clue to the discovery of the real nature of optic neuritis. It is interesting to know that at one time, impressed apparently by the association of speech defect with lesions of certain parts of the left hemisphere, he thought that defects of sight might be similarly associated with disease in a part of the right hemisphere [24]:—

"I have been struck with the greater frequency," he says, "with which I have found optic neuritis with hemiplegia on the left than on the right side. The facts I have observed go to prove that disease in a certain region of the left hemisphere produces loss of speech, and other facts go to show that disease in the corresponding part on the right does not affect speech at all. Having found disease here on the right with amaurosis several times, I have been led to pay much attention to this part, and I was at one time sliding into the belief that disease of it might be more likely to induce amaurosis than disease of the other parts of the right hemisphere."

A second point which emerges from a consideration of these papers is the importance he attached to ocular palsies in relation to vertigo. The study of this connected directly both with epilepsy and locomotor ataxy, and also with labyrinthine disturbances, but he dwelt on its importance in a wider sense, viz., because the facts of ocular vertigo are of great value in supporting from analogy the dictum that the anatomical substratum of a word is a motor, an articulatory process.

A third point which is to be noted is the remarkably high opinion which Jackson entertained of the educative effect of work carefully and energetically pursued at an ophthalmic hospital. He himself, as he repeatedly acknowledges, derived the greatest help from his early work at Moorfields, both because of the educative effect of the experience and the discipline in exact observation which it imposed. Such work, however, need not lead to a mere barren specialism or differentiation. As a matter of fact, it does not, for while ophthalmic studies have resulted in the increase in definite knowledge of eye conditions and diseases, they have also led to discoveries which have been a means of investigation of several important diseases, and they have been productive of the co-operation of neurologists and ophthalmic surgeons in the study and treatment of different morbid conditions. Such considerations obviously embody the evolutionary idea, and have in them the germ of the philosophical treatises on evolution and dissolution of the nervous system to which I have briefly referred.

I feel that I have not done justice to my subject. I doubt whether I could, if I were to elaborate it at much greater length. But I end on the note on which I began, for I trust that what I have said may open to some a new book, and may be to others a reintroduction to an old acquaintance. To me the study of these papers has been a source of intense interest and of great pleasure, and I have been anew impressed with the marvellous powers of one who was to many of us here a constant stimulus, and was besides, to me, a very dear friend.

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THE SENSORY FIELD OF THE FACIAL NERVE: A FURTHER CONTRIBUTION TO THE SYMPTOMATOLOGY OF THE GENICULATE GANGLION.[1]

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CHAPTER I.—INTRODUCTION.

AT a meeting of the American Neurological Association in May, 1907, a preliminary statement was made of my views regarding the general sensory system of the facial nerve and its clinical importance. This subject was still further elaborated in my presidential address to the New York Neurological Society in February, 1909 [18], and the attempt was made to outline the sensory system of the seventh cranial nerve, exclusive of its taste function and muscle sensibility, and to endow this system with a definite symptomatology similar to that of the other mixed cranial nerves. Thus the geniculate ganglion, its posterior root, the pars intermedia of Wrisberg and various peripheral divisions, were accorded a definite place in the realm of clinical neurology, supplementing the position which this system had long occupied morphologically in the domain of embryology and comparative anatomy (see fig. 1).

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[1] Amplification of a subject presented at the Seventh Annual Meeting of the American Society for the Advancement of Clinical Investigation, held in Washington, May 10, 1915.

The sensory system of the geniculate ganglion in man stands in close relation to the structures of the auditory mechanism, a natural sequence of its course and distribution in the lower vertebrate forms where it is the nerve of the first branchial or ear cleft. It participates in the innervation of the internal ear, the middle ear and its prolongation into the mastoid cells and Eustachian tube, as well as the skin of the auricle. There is also evidence to show the occasional presence of a vestigial innervation within the buccal cavity, on the palate and in

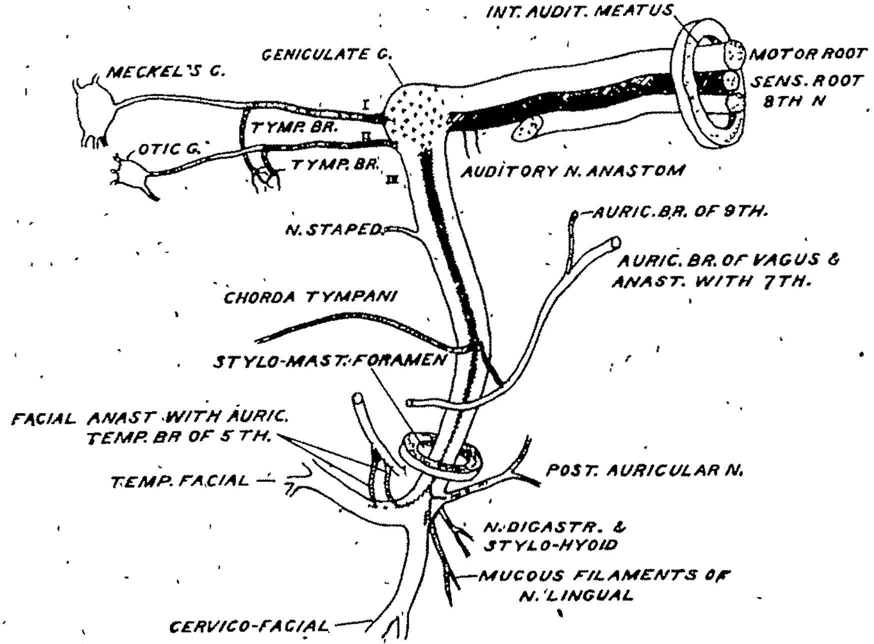

FIG. 1.—Diagrammatic representation of the facial nerve, showing the geniculate ganglion, its sensory root (the nerve of Wrisberg) and peripheral divisions.

1st Division—Great superficial petrosal nerve passing to Meckel's ganglion and its tympanic branch (great deep petrosal).

2nd Division—Small superficial petrosal nerve passing to the otic ganglion and its tympanic branch (small, deep petrosal).

3rd Division—The motor trunk, including the chorda tympani and sensory fibres to the external ear.

The sensory filaments in the motor trunk may reach the auricle by way of the auricular branch of the vagus: the posterior auricular nerve, by anastomoses of the temporo-facial with the auriculo-temporal branch of the fifth, or with the motor fibres destined for the minute intrinsic muscles of the external ear.

The lingual branch of the facial conveys mucous filaments to the anterior pillar of the fauces and adjacent palatal region.

the chorda distribution of the tongue, a remnant of the large intra-oral system which exists in fishes.

Among the more important symptoms which I referred to this sensory system are:—

(1) Various clinical forms of the geniculate neuralgia, viz., primary tic douloureux of the ear; reflex and post-herpetic otalgia; tabetic otalgia, associated with degeneration of the nerve of Wrisberg.

(2) Auditory and mastoid pains in cases of facial palsy (Fallopian neuritis), and in recent cases the occasional presence of hypæsthesia in the region of the concha auris.

(3) As a sensory pathway for the direct transmission of irritative stimuli to the facial nucleus, thus favouring the production of nuclear irritability with resultant facial twitchings, myokymia or spasm.

(4) Herpetic inflammations of the geniculate ganglion; a *syndrome* characterized by an eruption of herpes zoster on the central portions of the external ear, usually associated with facial palsy and frequently with auditory nerve symptoms: the acoustic disturbances when present varying in intensity from slight hypo-acousis to the severer forms of Ménière's disease.

In a later study [21] the special symptomatology of herpetic inflammations of the auditory, glosso-pharyngeal and vagal ganglia was also considered, and a provisional attempt was made to outline the various zoster zones of these ganglia both on the external ear and within the buccal cavity.

My object in approaching this field again is to review briefly some of the contributions which have been made by other observers since attention was first directed to the subject; to answer certain criticisms which have been directed against it, and to present the results of subsequent personal study and observations, more especially those pertaining to the cutaneous and intra-oral fields of the seventh nerve.

CHAPTER II.—A REVIEW OF RECENT CONTRIBUTIONS TO THE SENSORY FUNCTIONS OF THE FACIAL NERVE.

(A) *The geniculate neuralgia.*—The most important of the recent contributions to the symptomatology of the geniculate system is that by Clark and Taylor [2], in which a case of severe tic douloureux of the sensory system of the facial nerve was cured by intracranial section of its posterior root, the pars intermedia of Wrisberg. The pains were typically neuralgic in character, paroxysmal, lancinating, and very severe, and were localized chiefly in the depth of the ear, the anterior wall of the external meatus, and on a small area just in front of the ear (otalgia). Immediate and complete relief was obtained after section of the posterior root, the so-called physiological extirpation of the ganglion, a method advocated by Van Gehuchten and Spiller in cases of trigeminal neuralgia. This patient had been examined by a number of neurologists and all were agreed as to the true neuralgic character of the pain, and its limitations to the zone which I had outlined for the geniculate otalgia. Allen Starr [42], who had examined the patient independently, also reached the same conclusions and had advocated surgical interference similar to that which was carried out subsequently by Clark and Taylor. Through the courtesy of Dr. Clark and Dr. Taylor, I had several opportunities of examining this patient, and in my opinion the case was one of true geniculate neuralgia. Furthermore, it is of interest to record that the patient had been under observation at various times since the operation and only recently—i.e., six years after the root section—she was seen by Dr. George Crile, of Cleveland, who reported complete freedom from neuralgic pain in the ear. There has never at any time been a recurrence of the otalgia. One would, therefore, seem justified in accepting a case of this character and the brilliant result achieved by operation as a complete confirmation of views which I had previously expressed as to the sensory system of the seventh nerve, its relation to otalgia, the surgical possibilities offered, and that the geniculate neuralgia merits a definite place in the large clinical group of this affection [20 and 23].

Cases of a similar nature have apparently been encountered by Wilfred Harris [15] in his extensive experience with the treatment of trigeminal neuralgia by the method of alcohol injections. His comments on the subject of geniculate neuralgia are as follows: "Excluding those cases, not very uncommon, in which some neuralgia in front of the ear accompanies facial palsy or herpes within the auditory meatus, possibly due to geniculate inflammation, I have encountered two patients who suffered from almost identical paroxysmal neuralgia, which had lasted for ten and eleven years respectively, and in which the paroxysms of pain began with the throat or posterior palatal region on one side, and spread into the ear and in front of the ear on to the cheek, and down the side of the neck. The sudden onset and intensity of the pain and the recurrent paroxysms closely resemble chronic trigeminal neuralgia, but the distribution of the pain made me doubtful as to dependence on the fifth nerve, and in each case no alleviation was produced by successful injection of the third division of the fifth."

Charles K. Mills [30], in a critical discussion of the sensory functions attributed to the seventh nerve, opposes some of the views which I had advanced, and is not inclined to accept Clark and Taylor's case as one of pure geniculate neuralgia. He offers the suggestion that the pains may have been merely symptomatic and relieved by the decompressive effects of the operation, and not by section of the sensory root of the seventh. He would eliminate the case on these grounds as confirmatory of my views. It should be remembered, however, that this patient was observed not by one, but by a number of experienced neurologists (Allen Starr, Pierce Clark, Leszynsky, and Hunt), all of whom testify to the typical and strictly neuralgic character of the pain; the suggestion, therefore, that simple decompression could have affected a permanent cure in a case of this character—*intractable tic douloureux*—is, to put it mildly, a forced and unlikely hypothesis.

(B) *The sensory system of the seventh nerve and its relation to reflex racial twitchings and spasms.*—That the sensory system of the seventh nerve may be an important pathway by which irritating stimuli pass to the facial nucleus was emphasized in my original discussion of the symptomatology of this system. In this connection the Clark and Taylor case of geniculate neuralgia, already cited, is of considerable interest. At the presentation of this patient to the New York Neurological Society in May, 1910 [1], i.e., one year after the nerve had been divided, I called attention to the absence of the usual facial twitchings and spasms on the paralysed side. The patient had had originally a complete facial paralysis following division of the facial nerve, the division including its sensory root, the pars intermedia of Wrisberg. After the operation there had been gradual improvement and, at the time of the presentation, there was a very considerable return of power in the left side of the face, so that the eyelid could be closed and the angle of the mouth drawn to one side, and yet there was a complete absence of the contracture, with twitchings and over-action, which usually complicate the severer types of peripheral facial palsy. The absence of these motor phenomena in this case may be explained on the ground that the sensory root of the seventh had been divided, thus blocking the flow of afferent irritative stimuli to the nucleus. By this procedure the nucleus would be placed completely at rest as regards afferent stimuli through the facial system, and this may have had a bearing on the absence of the usual spasmodic manifestations. That section of a posterior root with consequent interruption of sensory stimuli may affect favourably spasmodic states and over-action of the motor neurones has been amply demonstrated by the results of Foerster's operation in spastic paralysis.

Only recently Lasarew [29] has emphasized the concurrence of paresis in one branch with contracture or over-action in one of the other branches of the facial in lesions which involve the nerve in the posterior fossa, especially neuro-fibromata of the acoustic. This phenomenon of paresis associated with contracture, as well as facial twitchings and myokymic quiverings, I have frequently observed in cases of acoustic tumour, and have thought that irritation of the sensory root of the facial may have been in part responsible for some of these peculiar phenomena—a theory which is more in harmony with such motor manifestations than would be that of distant compression of the nucleus through the medulla, or irritation of the motor portion of the nerve.

(C) *Herpetic inflammations of the geniculate ganglion.*—Since the publication of my monograph in 1910 on the "Herpetic Inflammations of the Geniculate, Auditory, Glosso-pharyngeal and Vagal Ganglia" [21] there have been a number of contributions to this subject, among the more important of which may be mentioned those of Claude and Schaeffer [4], Dombrowski [11], Closier [6], Dabney [8], Norman Sharpe [39], Dejerine, Tinel and Heuyer [10]. Cases of this affection have also been reported by Climenko [5], Sharpe [38], Muck [32], Hennebert [16], Halphen [13], Palmer [34], Mollison [31], Laignel-Lavastine [27] and Souques [41], the symptomatology corresponding to one or other of the various clinical forms of this affection.

Of special interest is the case reported by Claude and Schaeffer with herpes occipito-collaris et oticus, because of its severity, the evidences of multiple ganglionic involvement, and the large number of paralytic complications, the latter including unilateral paralysis of the intrinsic muscles of the eye, the abducens, the facial and auditory nerves. A case very similar in severity and extent of distribution was reported many years ago by Raynaud [36].

The case described by Dejerine, Tinel and Heuyer is a typical example of the geniculate syndrome, but has a special interest because of the wide extent of the anæsthetic area and the interpretation which it receives from these authors. In addition to anæsthesia in the conchal region, there was also demonstrable a hypæsthesia of the face and of the occipital region on the paralysed side, all of which was referred to the geniculate sensory area. This subject will receive special consideration in Chapter V.

Charles K. Mills [30] and Kidd [25] have both contributed critical papers in which my views regarding the sensory functions of the facial nerve have been challenged. Mills upholds the old traditional point of view, and would limit the sensory symptomatology of this nerve to the special sense of taste. He does not admit the existence of herpes zoster oticus of geniculate origin, but would interpret the eruption in such cases as trigeminal or cervical herpes, or, possibly, of glosso-pharyngeal-vagal origin. He does, however, accept the possibility of an inflammation of the geniculate ganglion in herpes zoster, but this would produce merely disturbances of taste and facial palsy, without herpetic eruption, and if this were present it would indicate an involvement of one of the other ganglia, viz., the fifth, ninth, tenth, or upper cervical.

Kidd, on the other hand, admits the probable existence of a visceral sensory function of the seventh nerve, and confines his criticism to the cutaneous representation on the auricle, the existence of which in man he strenuously denies. The answer to many of the objections which are brought forth by these writers will be found in my earlier contributions. It is, however, my purpose in this paper to consider in detail some of the questions which have been raised, and more especially that of the cutaneous zone, and to formulate more definitely some of my views regarding this subject, fortified as they are by subsequent experience and by contributions from other workers in this field.

CHAPTER III.—THE CUTANEOUS AND INTRA-ORAL ZONES OF THE GENICULATE GANGLION OUTLINED BY THE HERPES ZOSTER METHOD.

In my discussion of the distribution of fibres originating in the cells of the geniculate ganglion, it was shown that this sensory system bears a very close relation to the structures of the auditory mechanism, its filaments passing to the internal ear, the middle ear and its prolongations, and to the external ear. I will now consider in detail the cutaneous innervation on the external ear, for, like the fifth, ninth, and tenth nerves, the seventh has not only an intra-oral, but a cutaneous representation as well. And as the geniculate sensory field has been the subject of some controversy, as well as misapprehension, I will first review briefly my own views on this question, and then proceed to the more recent contributions of other writers bearing upon this question.

In my previous studies the attempt was made to outline the cutaneous zone of the facial nerve by the herpes zoster method, i.e., by the study and analysis of cases of pure herpes zoster oticus; in which the eruption was limited to small cutaneous areas on the external ear, corresponding in distribution to the geniculate, glosso-pharyngeal, or vagal ganglia. A detailed presentation of the various embryological, anatomical, and clinical facts which were gathered in support of this theory will be found in my earlier publications. For the present purpose it will be sufficient to quote certain of the conclusions which were reached in regard to the cutaneous zone of the seventh nerve [21].

"In eighteen of the cases (herpes oticus) the eruption was distributed in what I regard in part as the geniculate area on the external ear, i.e., the external meatus, concha, tragus, anti-tragus, anti-helix and fossa of the anti-helix, and the upper portion of the external surface of the lobule. This representation of the geniculate on the external ear is corroborated by the occasional presence of hypæsthesia in the region of the concha in cases of recent peripheral facial palsy. From the herpes zoster evidence I believe that we are justified in drawing the following conclusions. Pure herpes zoster oticus is dependent upon herpetic inflammation of the geniculate, glosso-pharyngeal and vagal ganglia. The ganglion of the tenth and the ganglion of the ninth are represented in part on the posterior portion of the tympanic membrane and auditory canal, as well as on the postero-mesial surface of the auricle and adjacent mastoid region. The zoster zone of the geniculate ganglion is represented on the external surface of the auricle, intercalated between the zone of the trigeminus in front and cervical ganglion behind. From the relation which the facial bears to the first visceral cleft, it is very probable that the zone of the geniculate also dips into the auditory canal as far as the tympanic membrane in the same manner as do the auditory strips of the trigeminus and glosso-pharyngeal vagus. As I have already indicated, a considerable allowance must be made in these zones, as I have outlined them for anatomical anomalies of various kinds and for the normal physiological variation and overlapping of sensory areas. Furthermore, these sensory areas are small, and, like the minute muscles of the external ear, must be regarded as more or less vestigial in character; in other words, as remnants of sensory zones which are gradually fading away under the overlap of larger and more important sensory systems."

The cutaneous zone of the facial nerve in this study was therefore localized on the central portions of the auricle, viz., the concha, the external meatus, tragus and anti-tragus, incisura intertragica, anti-helix and fossa of the anti-helix, and the upper portion of the external surface of the lobule.

The cutaneous zones of the glosso-pharyngeal and vagal ganglia were localized on the posterior portion of the tympanic membrane, the posterior wall of the auditory canal, together with a strip on the postero-mesial surface of the auricle and the adjacent mastoid region. In addition to their cutaneous zones, it was also shown that the glosso-pharyngeal and vagal ganglia have intra-oral fields as well, the herpes zoster pharyngis and the herpes zoster laryngis, respectively. For embryological reasons, and also because of occasional herpetic manifestations, it was thought that the geniculate was also represented within the auditory canal and on the tympanic membrane, thus sharing with the fifth, ninth and tenth nerves in the innervation of this region.

In the description of these auricular zones of the seventh, ninth and tenth ganglia, especial emphasis was placed upon their vestigial character and variability. They were regarded as sensory remnants which in the course of phylogenetic development are being replaced by the encroachments of the trigeminal and cervical areas. These facts seem to have been overlooked by some observers, and notably by Kidd [25] in his discussion of the geniculate cutaneous area.

Cases of herpes oticus in the geniculate zone	Auricle	Concha	External meatus	Tragus	Anti-tragus	Anti-helix	Fossa of anti-helix	Lobule	Post-auricular cleft	Tympanum	Auditory canal	Soft palate	Facial palsy	Acoustic symptoms
Hennebert	..	+	..	..	..	..	..	..	+	..	..	..	+	+
Idem	+	..	+	..	..	..	..	..	..	..	+	..	..	..
Idem	..	..	..	+	..	..	..	..	..	..	..	..	+	+
Muck	..	+	..	..	..	..	..	..	..	+	+	..	+	+
Dejerine	..	+	..	..	..	+	..	+	..	..	..	..	+	+
Mollison	..	..	+	..	..	..	..	..	..	..	..	..	+	+
Palmer	..	+	..	..	..	+	+	..	..	..	+	+	+	+
Closier	..	+	+	+	..	..	..	..	..	+	+	+	+	+
Climenko	..	+	+	..	..	..	..	..	..	..	..	..	+	+
Sharpe	..	+	+	..	..	..	..	..	..	..	..	..	+	+
Idem	..	..	..	..	+	..	..	..	..	..	..	..	+	..
Idem	..	+	..	..	..	..	..	..	..	..	..	..	+	..
Personal observations ..	..	+	+	..	..	+	+	..	+	..	..	..	+	+
Idem	..	+	..	..	..	..	..	..	..	..	..	..	+	+
Idem	..	+	..	..	..	..	..	..	..	..	..	..	+	+
Idem	..	+	+	..	..	.	..	..	..	..	..	..	+	..
Idem	..	+	..	..	..	.	..		..	..	..	..	+	+

The distribution of the eruption is indicated on the anatomical landmarks of the ear by +.

Since these conclusions were reached in 1910, twenty additional cases of isolated herpes zoster oticus are available for analysis in which the eruption was limited to certain small areas on the external ear, the trigeminal and cervical zones being free from eruptive manifestations. Of this number seventeen cases are to be regarded as occurring within the cutaneous geniculate zone. (See table, p. 426).

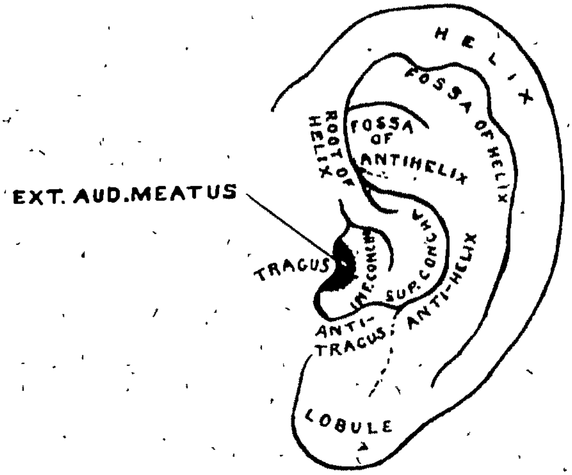

Fig. 2.—The topographical landmarks of the external ear.

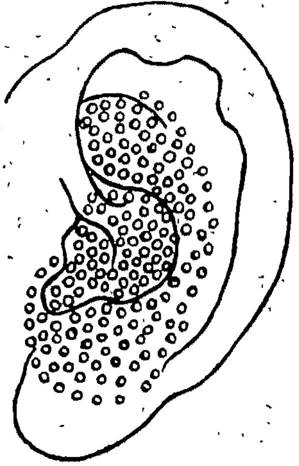

Fig. 3.—The cutaneous zone of the geniculate ganglion on the auricle, outlined by the herpes zoster method.

All of these were associated with a peripheral facial palsy, and in nine of the cases there were auditory disturbances as well. As will be seen by scanning the appended table, the eruptive manifestations in this series were distributed on one or more of the topograp marks of the external ear (see fig. 2), previously indicated as to the geniculate zone, viz., the concha and external meatı anti-tragus, incisura intertragica, anti-helix, fossa of the anti the superior portion of the lobule, the auditory canal and membrane (see fig. 3). I would, however, emphasize the f two of the cases, in addition to the typical geniculate erupti area, herpetic vesicles were also present in the cleft between t mesial surface of the ear and the adjacent mastoid, in a z corresponds to the innervation of the auricular branch of and which is a part of the vagal cutaneous zone; but, as the distribution of the glosso-pharyngeal vagus (herpes ph laryngis) in these cases was free from eruption, these retr

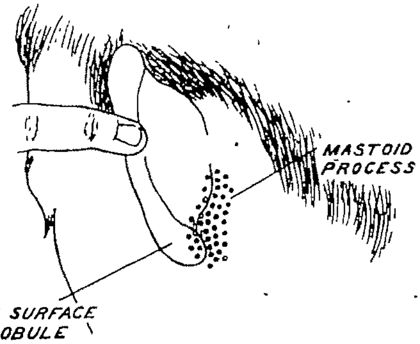

FIG 4.—The cutaneous strip of the geniculate ganglion on the postero mesi of the auricle, outlined by the herpes zoster method.

vesicles may be regarded as probably belonging to the genic In other words, the geniculate ganglion, in addition to i the central portion of the auricle, may in certain cases in the innervation of a strip of skin covering the pos surface of the auricle and adjacent mastoid (see figs. Another point of interest is that in two of the cases, in c with typical herpes in the geniculate area on the external was associated a unilateral eruption of vesicles within the c on the palate just in front of the anterior pillar of the fau confirms the existence of a visceral remnant of geniculate i within the buccal cavity, indicated in my previous comm In this relation the case reported by Frey [12] is especially i in which an eruption of herpes zoster was limited to the ton chorda tympani distribution, and like the geniculate cases was associated with facial palsy and auditory disturbances. Similar cases have also been described by Siegheim, Bernack, Eichhorst, Remak and Hunt [21], and suggest the possibility of common sensory fibres of geniculate origin passing to the tongue with the chorda tympani nerve.

To state the case briefly: the cutaneous zone of the geniculate corresponds in a general way to the concha auris and also adjacent marginal region, dipping into the auditory canal to the tympanum.

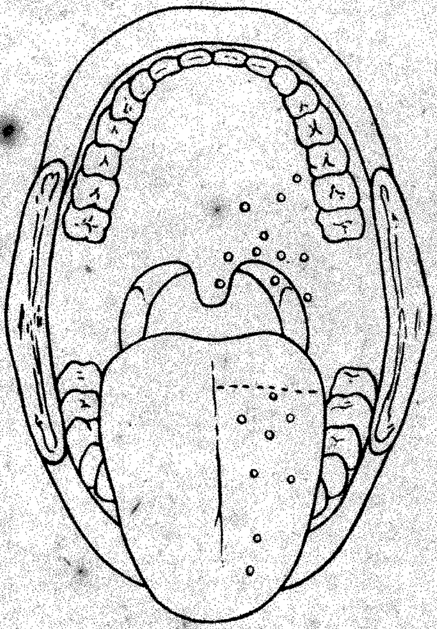

FIG. 5.—The distribution of intra-oral remnants of the geniculate visceral system on the palate and in the chorda distribution on the tongue, as determined by the herpes zoster method.

Its representation in this area is inconstant and variable. It has also an occasional, but quite inconstant, representation on the postero-mesial surface of the auricle and adjacent mastoid, thus sharing with the vagus in the innervation of this region. There is also clinical evidence in support of the occasional occurrence of an intra-oral remnant of geniculate origin on the palate and in the chorda distribution on the tongue (see fig. 5).

In reviewing the extent and distribution of the eruption in my first (eighteen cases) and second series (seventeen cases) of herpes oticus of geniculate origin, certain peculiarities are evident. This zone, so far as one is able to judge from herpetic manifestations, is small and variable. In certain cases only two or three small groups of vesicles may be present (see fig. 6). In other cases, the entire geniculate zone is fairly well covered, so that it is probable that the zone varies considerably in size and distribution in different individuals (see fig. 7).

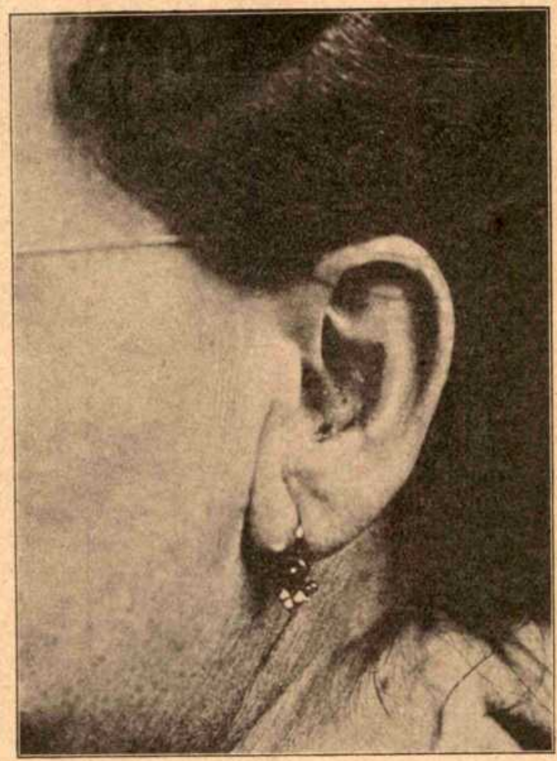

FIG. 6.—A case of herpetic inflammation of the geniculate ganglion with herpes oticus and facial palsy. Note the scattered groups of vesicles in the geniculate zoster zone; on the concha, root of helix, anti-helix, and the postero-mesial surface of the auricle; the trigeminal and cervical zones are free from eruption.

This is but natural in an innervation which is regressing phylogenetically, and in this respect is similar to the cutaneous zones of the ninth and tenth ganglia.

It is also significant that the geniculate zone should stand in such close relation morphologically to the small subcutaneous muscles of the auricle, viz., the muscles of the tragus, anti-tragus, and helix, which are themselves regressive and vestigial.

It might be suggested that the area outlined above for the geniculate really belongs to the glosso-pharyngeal-vagus innervation. Against this view may be mentioned the freedom from mucous membrane herpes in the intra-oral distribution of the ninth and tenth nerves and the absence of pharyngeal and laryngeal palsies, while a very important consideration in favour of the geniculate origin is the great frequency of facial palsy, a complication which was present in practically all of the cases, isolated herpes in the geniculate zone without facial palsy being very rare. It may, however, occur. It has been suggested by Kidd that herpetic involvement of the geniculate ganglion without facial palsy could not occur, and that therefore herpes zoster oticus without paralysis of the seventh nerve could not be of geniculate origin. While it is quite true that facial palsy does accompany herpetic involvement of the geniculate in nearly all cases, that it may occasionally escape involvement must be apparent to those who are familiar with the pathology and symptomatology

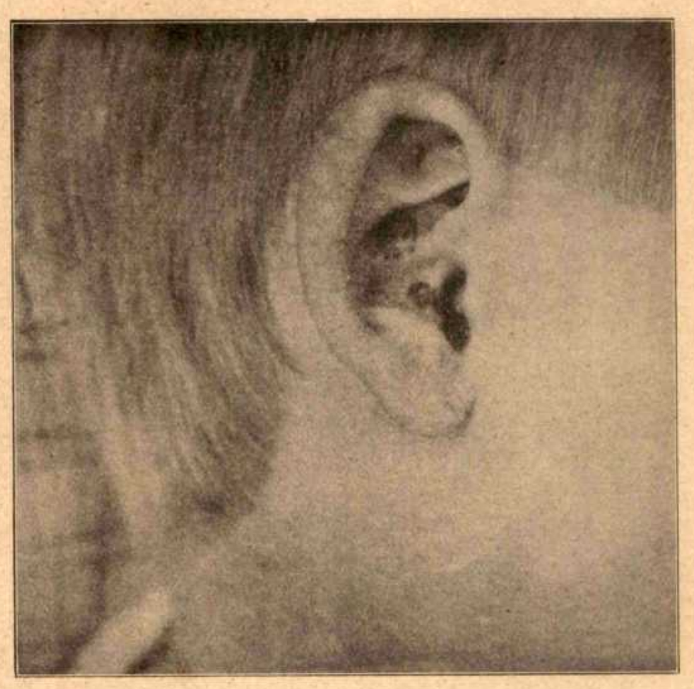

FIG. 7.—A case of herpetic inflammation of the geniculate ganglion with herpes oticus and facial palsy. The herpetic eruption is sharply limited to the geniculate zone on the auricle.

of herpes zoster. For example, in rare cases the inflammatory reaction in the affected ganglia may be very slight indeed, acute degenerative changes in the ganglion cells constituting the chief pathological lesion. As for clinical evidence, one has only to recall the isolated involvement of certain groups of cells in the Gasserian ganglion, with limitation of the eruption to only one of the branches of the fifth nerve, to realize that the inflammatory reaction within the ganglion may be extremely limited in extent. For these reasons, pathological and clinical, the occurrence of a herpetic inflammatory reaction in the geniculate ganglion with an eruption of herpes zoster and without paralysis of the adjacent motor nerve is not only possible, but probable.

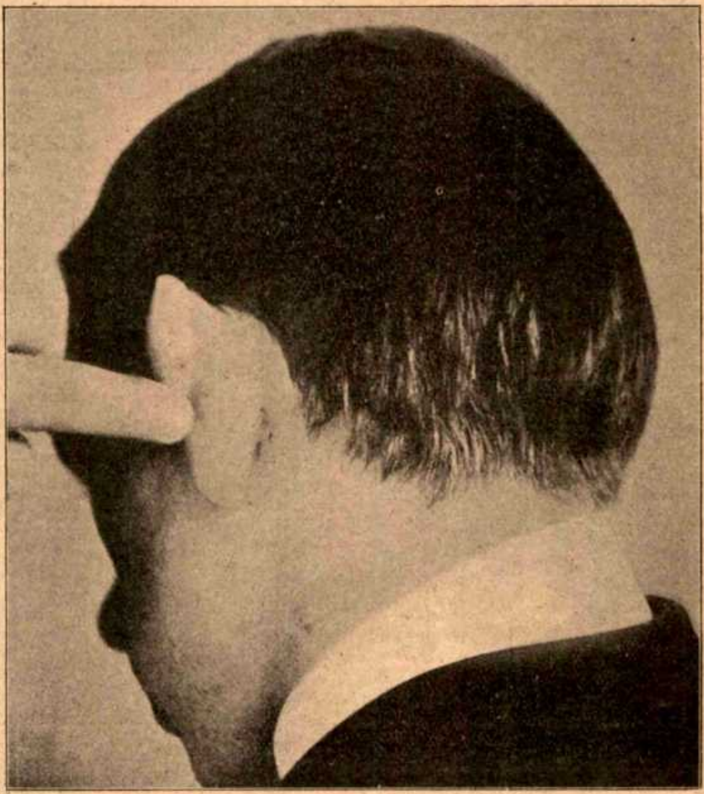

FIG. 8.—Herpetic inflammation of the geniculate ganglion with herpes oticus and facial palsy. The herpetic vesicles are limited to the posterior cleft between the auricle and mastoid.

CHAPTER IV.—THE PERIPHERAL INNERVATION OF THE AURICULAR AND INTRA-ORAL ZONES OF THE GENICULATE GANGLION.

A general outline of the auricular and intra-oral representation of the geniculate ganglion has been given as derived by the herpes zoster method. Particular emphasis was laid upon the variations in distribution as well as the vestigial character of this area. Attention was also directed to the intimate relationship and interlacement with neighbouring sensory zones of the fifth, ninth, tenth and upper cervical ganglia.

The cutaneous zone of the seventh, like the minute intrinsic muscles of the ear, is phylogenetically regressive, and is therefore subject in an unusual degree to anomalies of distribution. It is also significant that this cutaneous sensory remnant should correspond in distribution with those structures which are the outgrowth of the hyoid arch and the spiracle or ear cleft, which is, so to speak, the original domain of the seventh nerve. The external ear is developed from a series of small rounded prominences or auditory tubercles which originate in the mandibular and hyoid arches. The mandibular arch lies within the trigeminal area, while the hyoid arch is included within the area innervated by the auricular branches of the seventh, ninth, tenth and cervical nerves. As the seventh nerve is, properly speaking, the nerve of the first branchial cleft, its remnant of innervation in man represents the persistence of what in the lower forms was a larger and more important area.

The exact course pursued by the cutaneous fibres passing from the external ear to the geniculate ganglion is as yet unknown, and further experimental, anatomical and embryological investigations will be required in order to settle definitely this intricate question. I will, however, present my own views upon this subject, based on such facts as I have been able to gather, and while only tentative and requiring additional proof, they may be of assistance to those who approach the problem from another p[illegible]

On phylogenetic g[illegible]m probable that cutaneous fibres pass out with t[illegible]nerve and emerge with the motor fibres at [illegible]hat the facial at this (*a*) Through the communications which take place between the facial and the auricular branch of the vagus in the lower portion of the Fallopian canal.

(*b*) With the posterior auricular branch of the seventh nerve.

(*c*) Through communications between the main trunk of the seventh nerve and the auriculo-temporal branch of the fifth; and with the motor filaments destined for the innervation of the minute intrinsic muscles of the external ear.

(*a*) In "Quain's Anatomy" [35] the distribution of the auricular branch of the vagus is described as follows: "The auricular branch (nerve of Arnold) arises from the ganglion of the root (ganglion jugulare); it receives the filaments from the petrosal ganglion of the ninth; it then enters and traverses the substance of the temporal bone, crosses the aqueduct of Fallopius about 4 mm. from the lower end, forming a communication with the facial, and finally emerges between the external auditory meatus and the mastoid process. It divides into two parts, one of which joins the posterior auricular innervation of the seventh, while the other is distributed to the skin on the back of the pinna and the lower back parts of the auditory canal.[1] A similar distribution and connexion with the facial nerve is accorded the auricular branch of the vagus in "Testut's Anatomy." (See fig. 9.)

In the description of the collateral branches of the seventh nerve, Quain also states that "a communication with the auricular branch of the vagus is generally present in the form of a twig leaving the facial close above the stylo-mastoid foramen."

(*b*) The posterior auricular branch of the seventh nerve is given off as it emerges from the stylo-mastoid foramen. It then passes backwards and upwards to the anterior border of the mastoid process, and there divides into two branches—an ascending and a horizontal. The ascending or superior branch courses upward between the mastoid process and the auricle and terminates in the superior and posterior auricular muscles. The horizontal or posterior branch passes backwards and forwards and is [illegible] to the occipital muscle. Valentin [43] has describ[illegible]ts to the auricle from the ascending and hor[illegible]erior auricular branch of the facial nerve.

The sensory system of the geniculate ganglion has the following symptomatology:—

(*a*) Pain and sensory disturbances in facial neuritis.

(*b*) Neuralgic affections; primary tic douloureux, reflex otalgia, herpetic and tabetic otalgia.

(*c*) A sensory pathway for the direct transmission of irritative impulses to the facial nucleus, thus favouring the production of facial twitchings and spasms.

(*d*) Herpetic inflammations of the geniculate ganglion, a *syndrome* characterized by an eruption of herpes zoster in the geniculate zone, usually associated with facial palsy and often with auditory symptoms.

The cutaneous zone of the geniculate ganglion is represented on one or more of the following landmarks of the external ear (see figs. 2 and 3), viz., the concha, external meatus, tragus, anti-tragus, incisura intertragica, anti-helix, fossa of the anti-helix, superior portion of the external surface of the lobule, within the auditory canal and on the tympanic membrane. There is also reason to believe that the geniculate may in certain cases participate with the ninth and tenth ganglia in the innervation of a cutaneous strip on the postero-mesial surface of the auricle and adjacent mastoid region (see fig. 4).

In this outline of the cutaneous zone it is not to be assumed that this entire region is exclusively innervated by the geniculate ganglion, but merely that the geniculate zone which is variable in size and distribution falls within this area, and that other sensory systems, e.g., the fifth, ninth, tenth, and cervical nerves, also participate. Furthermore, as the cutaneous zones of the seventh, ninth, and tenth are rudimentary, there must be considerable differences in size and relationship in individual cases, and these variations in size and distribution, as well as the overlap of neighbouring systems, must be considered in the interpretation of their respective zones.

The same is true of the intra-oral remnant of the seventh nerve in the chorda distribution and in the region of the palate near the anterior pillar of the fauces, both of which are inconstant and variable (fig. 5).

The sensory disturbances following lesions of the facial nerve in in the Fallopian canal have the following characteristics: The pain is situated in the depths of the ear and mastoid region and in the central portions of the external ear, sometimes extending in the direction of the throat and tonsil. When severe, irradiation may also occur in the trigeminal, occipital and cervical areas. In some recent cases of facial palsy and occasionally after geniculate inflammations, a hypæsthesia in the region of the concha is demonstrable. It is slight and cannot be definitely delimited. Rarely in seventh palsies a postero-mesial strip on the auricle is also hypæsthetic. The inconstancy and moderate degree of the sensory disturbances when present are dependent upon the rudimentary nature of the sensory field of the geniculate, its variations in size, and its interlacement with the auricular branches of neighbouring distributions. For these reasons the herpes zoster method furnishes the best clinical criteria of its probable extent and variation.

It has been suggested that an eruption occurring within what I have described as the geniculate zoster zone may be of trigeminal, cervical, or glosso-pharyngeal vagal origin. That the occurrence of isolated herpes zoster oticus in this zone should be of either trigeminal or cervical origin is readily excluded by the small size and the peculiar location of the eruptive manifestations. Some of the cases are certainly of glosso-pharyngeal and vagal origin, and elsewhere the attempt has been made to isolate and separate their zones from those of geniculate origin. That the other cases are certainly of geniculate origin is shown by the almost constant association of facial palsy due to the proximity of the motor fibres of the seventh to the affected ganglion. Were these cases of glosso-pharyngeal or vagal origin, facial palsy should not occur so constantly. One would anticipate rather pharyngeal and laryngeal palsies or symptoms of vagal irritation (McKenzie [48]). Indeed it would be in just such lesions of the vagal ganglion that one would expect symptoms of pneumogastric irritation, e.g., coughing, singultus, bradycardia, nausea, and vomiting, and yet these distinctively pneumogastric symptoms were absent in the thirty-five cases of pure herpes oticus which I analysed in differentiating the geniculate zone.

As my results are based very largely on the analysis and study of cases by the "herpes zoster method," they cannot be accepted as the final solution of this difficult problem. Many more observations will be required in order to determine more accurately the extent of these auricular and intra-oral zones and their relationship, as well as further experimental and morphological studies.

It is, however, a beginning and may serve as a guide in future investigations.

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BILATERAL LESION OF THE OCCIPITAL LOBES WITH RETENTION OF MACULAR AS DISTINCT FROM PANORAMIC VISION.

CLINICAL RECORD

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HISTO-PATHOLOGICAL RECORD

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AND

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CLINICAL RECORD.

THE patient, a man, aged 45, was sent to me (B. B.) by Dr. (now Sir) George A. Berry in February, 1886, complaining of loss of sight. Dr. Berry reported that there was nothing wrong with his fundus. The loss of sight, which consisted of complete loss of peripheral vision with very marked reduction of macular vision, had come on suddenly with an attack of what appeared to be uræmic convulsions in June, 1884.

I diagnosed the condition as probably due to a bilateral lesion of the occipital lobes in the region of the half-vision centre. The case did not appear to be one of the rare cases of double homonymous hemianopsia, in which a lesion of one occipital lobe (say the right) produces a homonymous hemianopsia on the opposite (say the left) side, and a second and subsequent lesion in the other (say the left) occipital lobe produces a homonymous hemianopsia on the opposite (say the right) side, but a *simultaneous* bilateral lesion in the right and left occipital lobes—an infinitely rare, if not a unique, condition.

I recorded the case in my "Atlas of Clinical Medicine" (vol. i, p. 49), published in the year 1893 (seventeen years before the patient's death), and there stated :—

"This is a most interesting case. In the absence of a post-mortem examination it is, of course, difficult to say with any degree of certainty what is the position and pathological character of the lesion. But the condition of the visual defect—complete abolition of peripheral vision, with, comparatively speaking, good central vision both for white and for colours—and the absence of any changes in the fundus oculi are, I think, strongly suggestive of a bilateral lesion of the half-vision centre in the back part of each occipital lobe."

I watched the patient carefully until his death on October 21, 1910, a period of twenty-four and a half years, making it worth his while to see me every six months, in order that I might keep in touch with his condition. During the whole period (twenty-four and a half years) that the patient was under my observation the condition remained, practically speaking, unchanged, and there were no further cerebral developments; central (macular) vision improved slightly, peripheral vision remained completely obliterated. Throughout the whole course of the case there was no motor paralysis, no loss of sensation, and no aphasia, either sensory or motor.

In April, 1905 (twenty-one years after the onset of the lesion, and five years before the patient's death) Dr. A. H. H. Sinclair kindly examined the patient and gave me an exhaustive report on the condition of vision and of the fundi, and accurate charts of the fields of vision (figs. 3 and 4).

The patient died of acute croupous pneumonia on October 21, 1910, aged 71. Through the kindness of Drs. George Donald and George M. Johnston, of Leith, I was able to make a post-mortem examination.

A large lesion was found in each occipital lobe.

The case seemed to me to be of so much scientific interest that I asked Dr. J. Shaw Bolton to undertake the microscopical examination. This he very kindly consented to do.

It is worthy of note, considering the great extent of the lesion in both occipital lobes and the extensive severing of the connexions between the visuo-sensory centre and the other parts of the brain, that there was no mind-blindness.

The case is, as Dr. Shaw Bolton states, unique and of the highest scientific value. It was carefully studied during life and watched for a period of twenty-four and a half years by myself; the ocular and visual condition was carefully noted by two leading authorities on ophthalmic medicine—at the commencement of the case by Dr. (now Sir) George A. Berry and at the end of the case by Dr. A. H. H. Sinclair; and the histological investigation, involving as it has done an enormous amount of time and labour, was carried through by the leading authority on the histology of the visual area and occipital lobe, Dr. J. Shaw Bolton, assisted by Dr. William Robinson.

The details of the case are as follows :—

Acute general dropsy (probably due to acute nephritis); epileptiform convulsions; permanent loss of peripheral vision, the fields being enormously contracted; central vision both for white and for colours fairly good; fundi oculi normal; no mind-blindness and no word-blindness. The diagnosis, made twenty-four years before death, was "probably a bilateral lesion in the occipital lobes"; death at the age of 71, twenty-six and a half years after the onset, from acute croupous pneumonia. During these twenty-six and a half years there were no fresh head symptoms, and practically no change in vision, no motor paralysis, no loss of sensation, no aphasia, sensory or motor. On post-mortem examination a large lesion was found in each occipital lobe.

M. M'G., a labourer, aged 45, was sent to me on February 24, 1886, complaining of loss of sight.

Previous history.—The patient stated that he had enjoyed good health until June, 1884, when he had a severe illness, which seemed to have been acute nephritis with uræmia. The attack began with bronchitis and "stoppage of the water" (he does not know if the water was red or bloody), his face, body, and limbs swelled, and he lost his appetite. During the course of the attack he suddenly lost his eyesight, and almost immediately afterwards had a series of epileptic fits. He was unconscious for fourteen days; it was three weeks before he could recognize voices. When he regained consciousness he was quite blind; since then his eyesight (central vision) has been gradually restored in some degree.

Since he recovered from the acute stage of the attack his general health has been good; he now feels well, if it were not for the loss of sight; he is, however, "at times heavy and inclined to sleep" For some time after he recovered from the acute attack he occasionally suffered from headache, but the pain was not frequent and not severe; for the past year there have been no headaches. He has not suffered from giddiness or vomiting. His face has never swelled since he recovered from the acute attack. He has not had syphilis.

Condition on February 24, 1886.—The urine was absolutely normal, and the heart and other organs quite healthy; the patient was muscular and well nourished. There was no motor paralysis, no loss of sensation, and no aphasia, either sensory or motor.

The knee-jerks were normal; there had been no lightning pain; the pupils were of medium size, they contracted actively both to light and accommodation—in short, there was no suspicion of tabes.

Central vision for white was very defective (V. = $\frac{3}{18}$). The peripheral portions of the field were so greatly constricted that peripheral vision was *nil.*

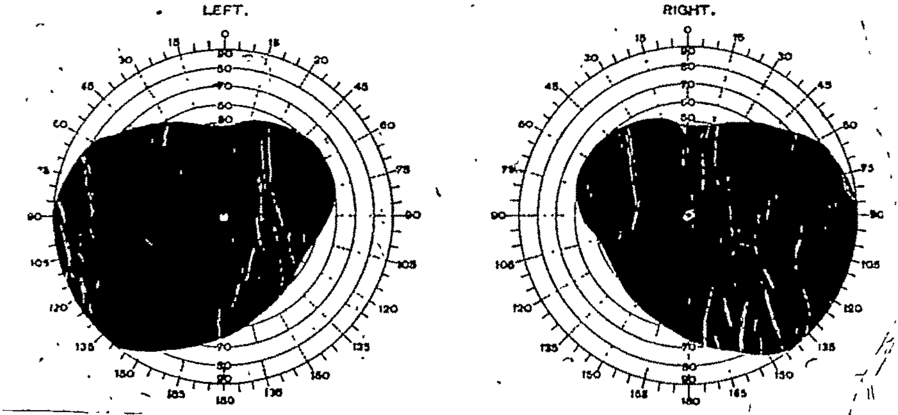


FIG. 1.—Perimeter chart of the fields of vision in the case of M. M'G., described in the text, taken on January 12, 1887. (Reduced from 9½ in. to 4½ in.)

There were no ophthalmic changes—a fact confirmed by Dr. Berry.

Subsequent progress of the case (*January* 12, 1887).—The patient's general health is good. His sight is better; he says that he can see little objects, such as a pin on the floor, but that he fails to see larger objects, and often runs up against things, tumbles over his children, &c. Central vision has improved—right eye = $\frac{5}{18}$ imperfectly; left eye = $\frac{3}{18}$ imperfectly; central colour vision good. The condition of the fields at this date (January 12, 1887) is shown in fig. 1.

March 19, 1891.—His general health is good. The urine continues quite normal; there have been no head symptoms—in fact, no nervous symptoms at all, except the affection of sight. He stated that he sees objects distinctly when they are placed directly in front of him; and that he can see small objects, such as a pin or a small piece of white paper the size of a pea on the floor. I satisfied myself that this statement was correct. In order to see a small object on the floor in front of him he moves his head and eyes about (ranges over the area where he knows the object is) until he gets it in the line of central vision.

Central vision is fairly good—much better than it was four years ago; at 18 ft. he correctly named with the right eye one of the test letters which ought to be read at 18 ft., and three out of five of the letters which ought to be read at 24 ft. (Right eye, V. = $\frac{18}{24}$ imperfectly.)

With the left eye he could not recognize any of the letters which ought to be read at 18 ft., but at this distance he correctly named three of the five letters which ought to be read at 24 ft. (Left eye V. = $\frac{18}{24}$ imperfectly.)

Vision was not improved by glasses.

Central vision for colours was perfect in each eye.

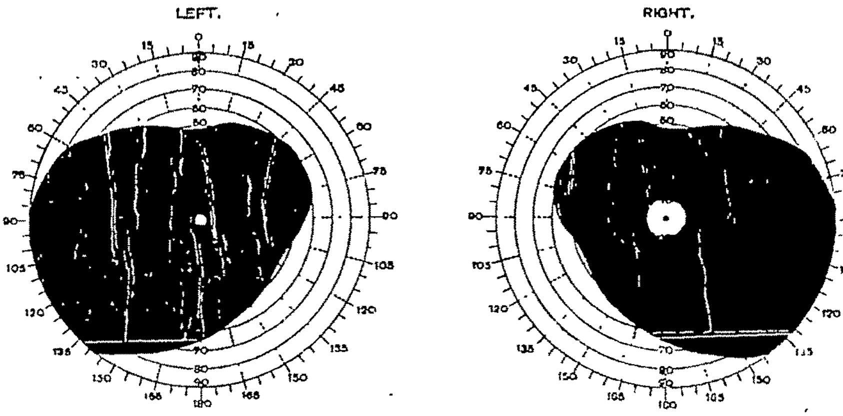


FIG. 2.—Perimeter chart of the fields of vision in the case of M. M'G., described in the text, taken on March 19, 1891. (Reduced from 9¼ in. to 4½ in.)

He says he cannot see anything at the sides, above or below. The fields of vision were again mapped out with the greatest care; the area of central vision was a little, but only very little, larger than on January 12, 1887; the improvement was most marked in the right eye (see fig. 2).

The pupils contracted actively both to light and on convergence.

There were no abnormal ophthalmoscopic appearances.

The day on which these observations were made was particularly clear and bright.

His mental condition is not very bright, and he says his memory is not good, but there is no mind-blindness and no word-blindness. walks about the streets without assistance, coming up every six by himself to the Royal Infirmary to see me. He can read larg such as the prices of objects in shop windows.

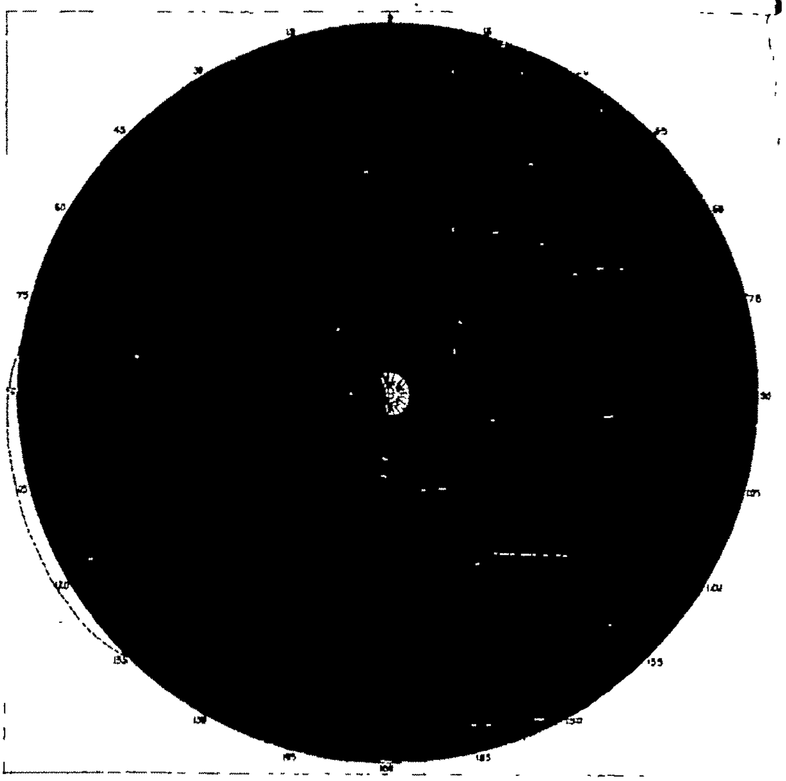
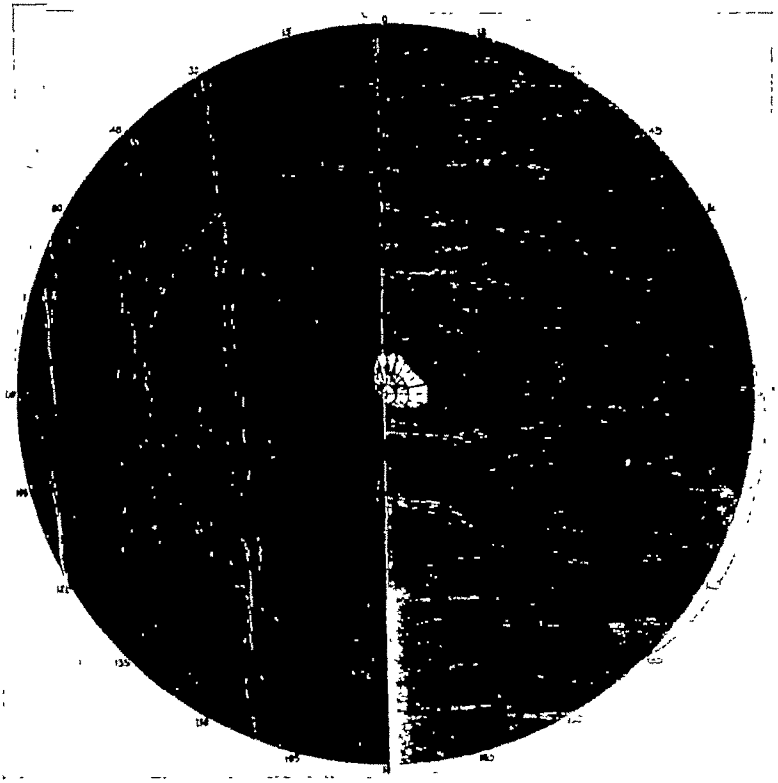


FIG. 3.—Perimeter chart of the *left* field of vision in the case of M. M'G., taken Dr. Sinclair on April 10, 1905. (Reduced from 7½ in. to 4 in.)

The dark interrupted line encloses the area over which the field of vision normally ext The part of the dark shading within the dotted line shows the extent to which blindnes encroached on the field, and the area at the centre, which is very lightly shaded, indicate remaining field. The remaining field responded to a white test object 10 mm. in dia and seen at 250 mm. distance (as taken with the perimeter). The small white areas w the remaining field on the right side of the fixation point responded to a smaller stimulus a white test object 10 mm. in diameter and seen at 2,000 mm. distance on Bjerrum's scre

On May 19, 1891, the patient was admitted to the Edinbu Royal Infirmary suffering from scurvy, under Professor Wyllie, has kindly allowed me to see his notes. The attack had commen twelve days previously, and had evidently produced marked (tempor deterioration of vision and of the mental condition. The patient was stupid, his memory was poor, the vision very defective. He was discharged cured of the scorbutus on June 18, 1891.

January 8, 1893.—There is no change in the general condition vision is not so good as it was on March 9, 1891.



IG. 4.—Perimeter chart of the *right* field of vision in the case of M. M'G , taken by Dr. Sinclair on April 10, 1905. (Reduced from 7½ in. to 4 in.)

The dark interrupted line encloses the area over which the field of vision normally extends. e part of the dark shading within the dotted line shows the extent to which blindness had roached on the field, and the area at the centre, which is very lightly shaded, indicates the aining field. The remaining field responded to a white test object 10 mm in diameter l seen at 250 mm. distance (as taken with the perimeter). The small white areas within e remaining field on the right side of the fixation point responded to a smaller stimulus, i e., white test object 10 mm. in diameter and seen at 2,000 mm. distance on Bjerrum's screen.

April 26, 1894.—Does not feel so well;—thinks his sight is not so good; is occasionally giddy; a strange feeling—"sort of weakness"—omes over him at times. His memory, he says, is impaired, but his visual memory (for places, objects, &c.) is not specially interfered with.

He walked up alone from Leith to the Edinburgh Royal Infirmary to-day.

April 8, 1905.—*In statu quo.* The patient continues to comes up to the Infirmary to see me every six months, as he has done regularly during the past twenty years. General health good.

Dr. Sinclair kindly made an exhaustive examination, and sent me the following report on the ocular condition and vision:—

(*Dr. A. H. H. Sinclair's Report, April* 10, 1905.)

There is some slight chronic conjunctivitis present in both eyes—right eye, V. = $\frac{6}{36}$ uncertainly; left eye, V. = $\frac{6}{36}$ and $\frac{6}{24}$ partly. Central fixation is present, but is not perfect.

The field of vision is very greatly contracted, and more so on the left side in both eyes (see figs. 3 and 4). The fixation point is probably encroached upon on the left side in both eyes. The slightly larger extent of the right field corresponds to the patient's statement that he can see "a little wider with the right."

The pupils are small (3 mm.) react to light, and are equal.

The eyes move normally. Convergence and other movements are uncertainly performed, because of the failure in indirect vision.

The *ophthalmoscopic examination* shows both discs to have a somewhat leaden-grey atrophic appearance, with extensive shallow cupping probably more than the original physiological cupping.

There is a well-developed scleral ring round each disc—quite like the high-water mark seen round the disc in glaucoma; though not in this case connected with glaucoma, it is an evidence of the atrophic process which apparently has been going on for a long time.

The general fundus shows in each case some tessellation of the choroid. In the left eye there are some slight changes in the choroid between the disc and the macula.

The retinal arteries have a hard "silver-wire" look, but otherwise the retinal vessels are normal.

On June 15, 1905, the patient was admitted to the Edinburgh Royal Infirmary, under my care, suffering from scurvy of a fortnight's duration. He was discharged cured of the scorbutus on July 7, 1905.

June 2, 1910.—No change since the last note; general health good; walked up from Leith alone as usual to see me to-day; his memory, he says, is no worse; can read large type; can write, but does not write letters—his sight is so bad that the lines run into one another.

After the patient's death his daughter, with whom he lived, told me that she remembered his having one convulsion of recent years. He could, she said, read large type, and could tell the prices of objects in the shops. He only once, so far as she remembered, wrote a letter; it was very bad, for he wrote one line on the top of another. He never complained of his head; his memory was good, and his mind was all right. He was able to find his way about quite well.

The patient died, at the age of 71, from acute croupous pneumonia on October 21, 1910. Through the kindness of Drs. Donald and Johnston I was informed of the patient's death, and was able to get permission for a post-mortem examination.

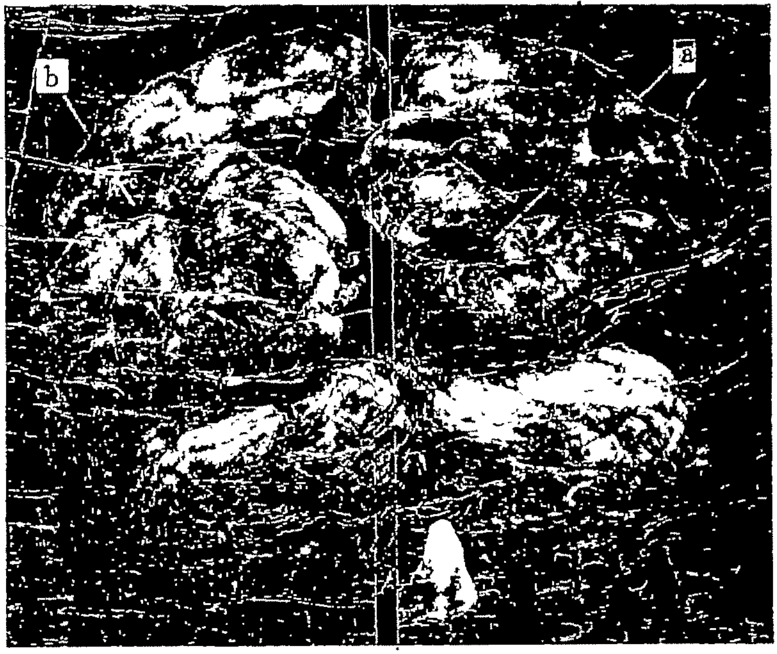


FIG. 5.—Brain in the case of M. M'G., showing the appearance of the occipital lobe as seen from behind. The letter "a" points to the lesion in the right, and the letter "b" to the lesion in the left occipital lobe.

Post-mortem Examination.

This was made on October 22, 1910 (twenty-six years after the occurrence of the cerebral lesion), by Mr. W. Waldie in my presence.

Head.—The dura mater was somewhat thickened and adherent at the vertex.

The arteries at the base of the brain were somewhat atheromatous; a definite patch of atheroma was situated in the right posterior cerebral artery just beyond its junction with the circle of Willis, and another on the left common carotid artery just at its junction with the circle of Willis.

The convolutions of the brain, more particularly of the frontal lobes, were somewhat atrophied and separated by deep sulci—apparently a senile change.

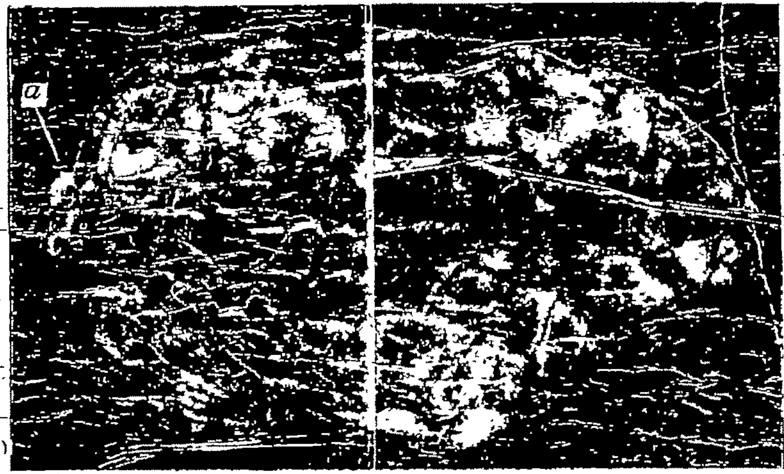


Fig. 6.—Lateral view of the right side of the brain in the case of M. M'G., showing the appearance of the occipital lobe, to which the letter *a* points.

A large lesion was present in each occipital lobe and a small superficial lesion in the adjacent part of the right temporo-sphenoidal lobe (posterior end of the middle temporo-sphenoidal convolution).

In the right occipital lobe an extensive lesion was seen (*see* figs. 5 and 6), a marked depression in the middle of the lobe, and shrinking in of the convolutions, with discoloration, involving more particularly the upper and middle convolutions; the lower convolution at its tip and the visual area (cuneus and calcarine area) appeared to be normal.

In the left occipital lobe there was a marked depression on the upper surface, apparently leading into a cavity in the interior of the brain (*see* figs. 5 and 7). The surrounding convolutions were plump and apparently normal.

Viewed from the side, there was marked depression at the junction of the right occipital lobe with the back part of the temporo-sphenoidal lobe (*see* fig. 6). A small part of the posterior end of the right middle temporo-sphenoidal convolution was shrunken and atrophied (the result of an old softening).

There was also an area of superficial softening in the middle part of the middle and lower temporo-sphenoidal convolutions on the left side (*see* fig. 7): it was separated from the occipital lobe by an area of normal brain tissue, measuring 1 in. from before backwards.

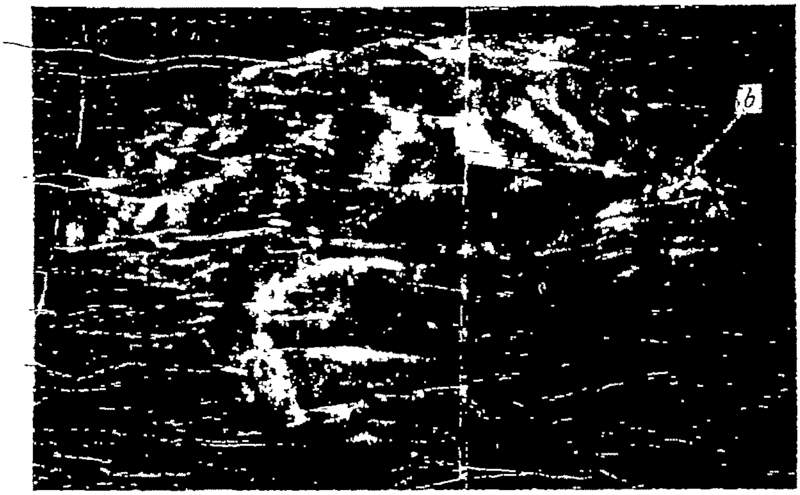


FIG. 7.—Lateral view of the left side of the brain in the case of M. M'G. showing the appearance of the occipital lobe, to which the letter *b* points.

After separating the cerebellum and looking at the back part of the base of the brain from below, there was a marked depression on the right side at the junction of the occipital and temporo-sphenoidal lobes. The convolutions of the occipital lobes (both right and left) on their under surfaces were plump and normal.

The brain was injected with 10 per cent. formalin. On November 12, 1910, it was cut into three sections (*see* fig. 8). The lateral ventricles were greatly dilated. The whole brain was sent to Dr. Shaw Bolton for further examination.

The brain lesion consisted in extensive destruction of the white matter and part of the cortex of the left occipital lobe and very extensive destruction of the white and grey matter of the right occipital lobe. The lesions apparently were the result of old softenings, probably, from the very sudden onset, embolic in origin, with great secondary (compensatory) dilation of the lateral ventricles

Lungs.—*Right*—the upper lobe, which was adherent, was consolidated by acute croupous pneumonia. On section, the affected area was found to be in a state of grey hepatization, almost gangrenous. *Left*—congested and œdematous, but otherwise normal.

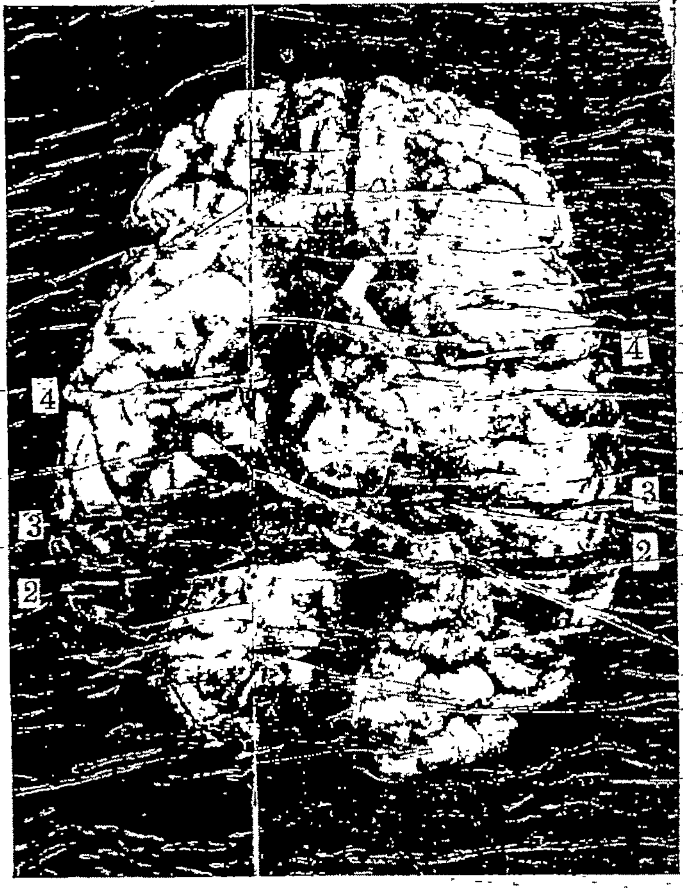


FIG. 8.—Base of cerebrum showing the positions of the transverse vertical sections 2, 2, 3, 3, and 4, 4, the front faces of which are respectively illustrated in figs. 12, 10 and 9. The back face of section 2, 2 is shown in fig. 13. R, cuneus of right hemisphere, which, owing to the lesion, in this position of the cerebrum looks upwards instead of inwards.

Heart.—This showed senile changes but no valvular lesion.

Kidneys.—Both were rather pale and somewhat atrophied, but not markedly abnormal.

Bladder.—This was much thickened and encrusted with phosphates—acute and chronic cystitis.

HISTO-PATHOLOGICAL REPORT.

General naked-eye appearances. — The base of the brain shows but one abnormality, namely, a partial rotation of the right occipital pole, which results in the right cuneus (fig. 8, R) looking downwards and inwards instead of inwards. This, as will be seen, is due to the lesion in the right occipital lobe. Sections (front face) through lines 4, 4, and 3, 3. in fig. 8 are shown respectively on figs. 9 and 10. These sections call for no special remark. The lateral ventricles are dilated and there is some degree of lack of symmetry, but even the latter of these figures is anterior to the limits of the lesions.

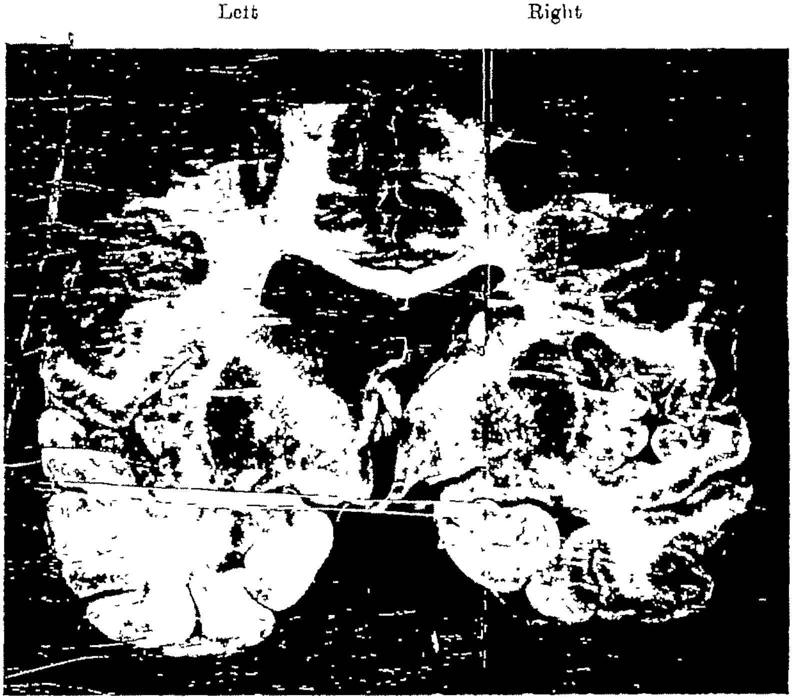


FIG. 9.—Front face of transverse vertical section 4, 4 of the preceding figure. The right lateral ventricle looks smaller than the left both in this figure and in figs. 10 and 12. This is due to the larger size of the lesion on the right side with the greater consequent loss of cerebral tissue and secondary shrinkage which have resulted No actual lesion can be seen in this figure.

The optic nerves, basal ganglia, &c., are to the naked eye normal.

The lesions.—These are shown from behind in fig. 11. The left lesion on the stripped specimen is merely a deep fissure or scar on the outer surface of the occipital lobe, and has associated with it a

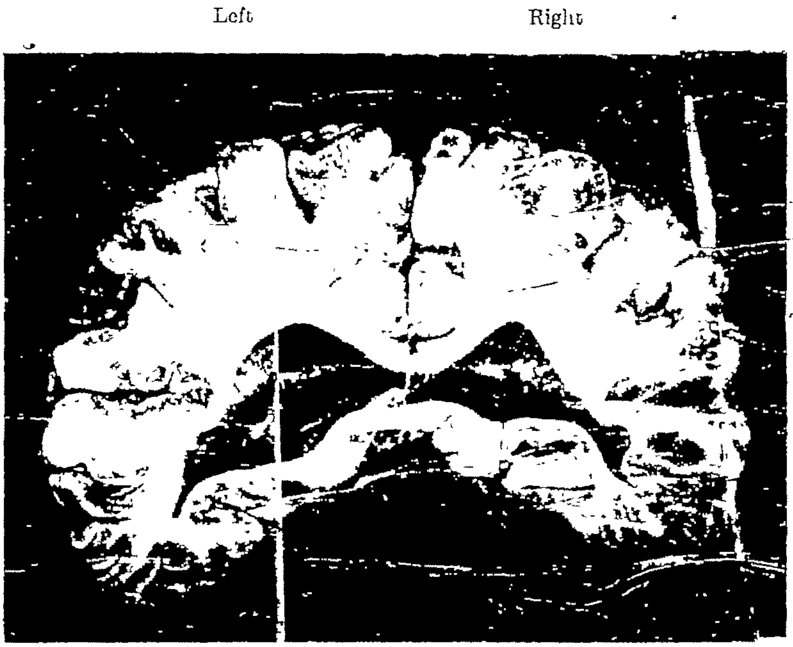


FIG. 10.—Front face of transverse vertical section 3, 3 of fig. 8. The remarks in the legend to the preceding figure apply equally to the present figure.

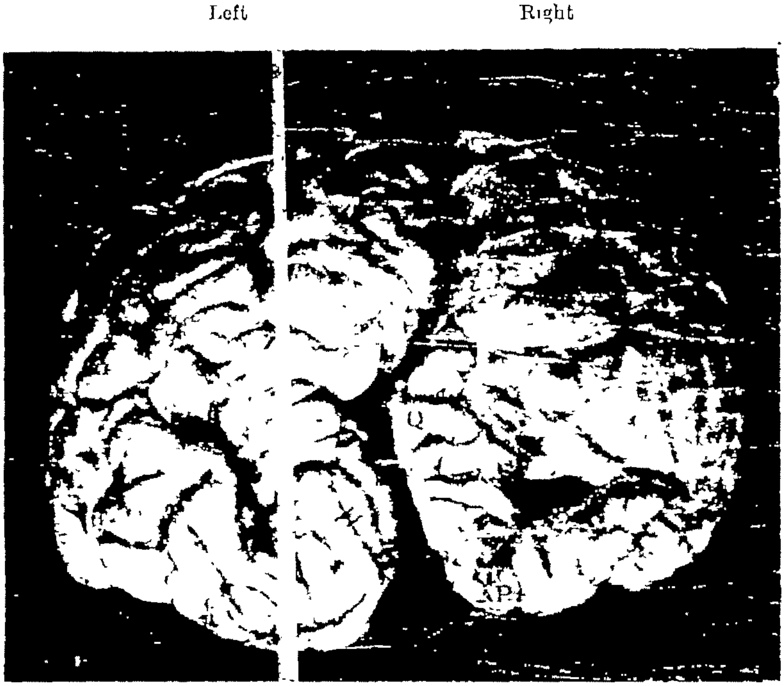


FIG. 11.—Cerebrum from behind, showing the almost vertical left-sided lesion as a deep scar, and the large triradiate scar on the right side. Q, right quadrate lobule, P, pole of right hemisphere; =, upper end of the radiate extremity of the left calcarine fissure. The floor of the extensive right-sided lesion is not visible in this figure. It is shown in figs. 12 and 13 respectively in its natural shrunken and in the stretched state.

folding inwards of the quadrate lobule and the cuneus to an obtuse angle with one another. The right lesion is a much more extensive triradiate scar extending to the pole (fig 11, P). This scar, in its natural folded-up state, results in a <-shaped projection mesially (fig. 11, Q), of which the upper limb and angle are the quadrate lobule and the lower the cuneus. This projection fits into the depression between the left quadrate lobule and the cuneus. The long posterior extremity of the left calcarine fissure is marked on the figure by a =.

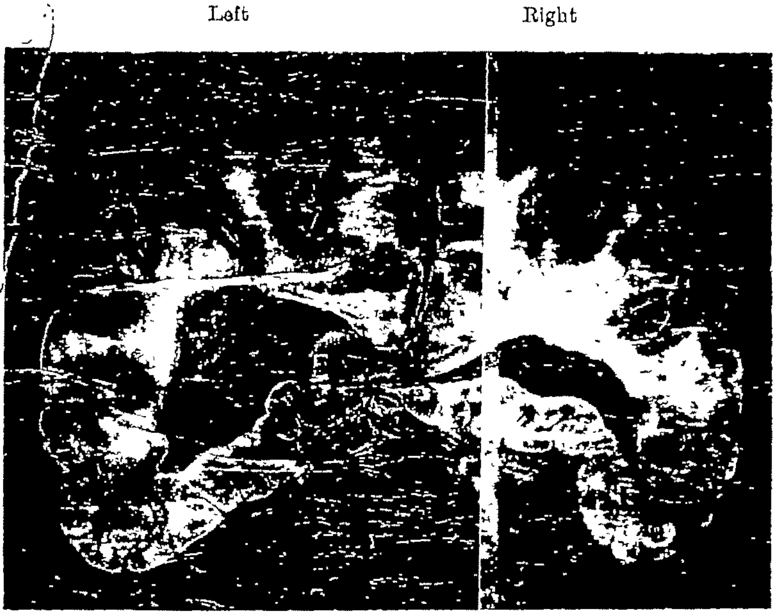


Fig. 12.—Front face of transverse vertical section 2, 2 of fig. 8. This figure shows that the lateral branch of the triradiate lesion of the right side extends forwards to beyond the plane of the section. The numerals 1 and 2 are respectively placed above and below the lesion, the shrunken floor of which is seen between them. As the consequence of this shrinkage the right hemisphere and the right ventricle look smaller than the left hemisphere and the left ventricle. The first of these appearances is real the second, as the following figure shows, is illusory.

The lateral limb of the triradiate right-sided lesion extends forwards for some distance beyond the line 2, 2, on fig. 8. This is indicated by the numerals 1 and 2 on fig. 12, which represents the anterior face of section 2, 2 on fig. 8, and by the same figures on fig. 13, which shows the posterior face of the section 2, 2 on fig. 8. In the latter figure, the posterior horns of the respective right and left lateral ventricles are filled with cotton-wool, and thus the extensive floor 1, 2 of the lesion of the right occipital lobe is exposed. The floor of this lesion, when opened out, occupies an area of about 4 sq. in. This lesion, as stated, extends forwards by its lateral limb beyond the line 2, 2 of fig. 8, and by its postero-inferior limb (fig. 11, P) it reaches the pole of the right hemisphere and nearly

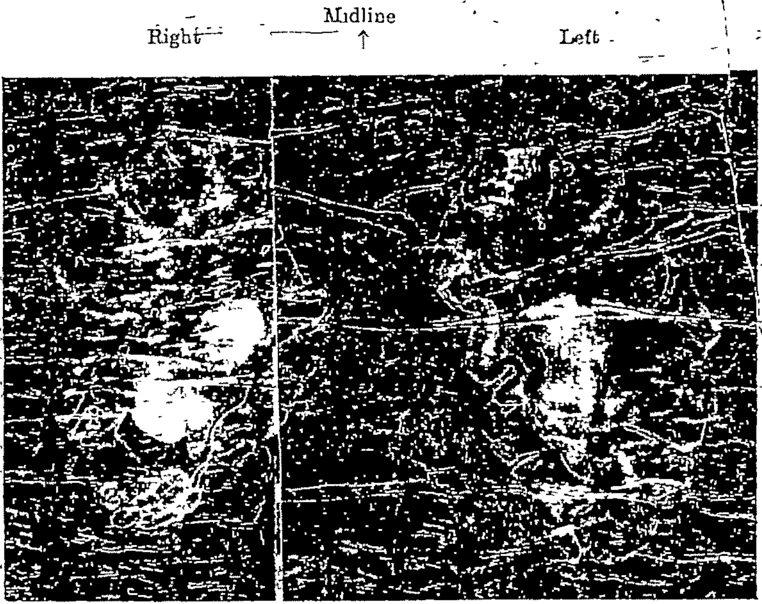


FIG. 13.—Back face of transverse vertical section 2, 2 of fig 8. This figure seen in a looking-glass becomes the duplicate of fig. 12. The ventricles are filled with cotton-wool. The right ventricle is here seen to be the larger, and between numerals 1 and 2 the floor of the lesion, which consists solely of pia-arachnoid and ependyma, is well shown. It is owing to the great loss of tissue caused by the right lesion that the right cuneus looks downwards instead of inwards when the brain stands in its natural position. (fig. 8, R), and that the quadrate lobule projects so markedly inwards (fig. 11, Q).

to the outer extremity of the calcarine fissure of this side. *Nowhere else in either hemisphere* (as will be shown later) *does the lesion come into even the approximate neighbourhood of the visuo-sensory area.*

The visuo-sensory areas and the fissural pattern of the calcarine region of the occipital lobes.—The visuo-sensory areas of the two occipital lobes have been carefully delimited histologically by the method of piecemeal examination, which, though very laborious, is the only really satisfactory one, and a description of these areas will now be given. The necessary account of the fissural pattern of the needed portions of the respective occipital lobes will be included in this description, as it would serve no useful purpose to give these separately.

As, however, the convolutional patterns of the two sides, as well as the size and shape of the respective visuo-sensory areas, present important differences, it is necessary to deal with these sides quite separately.

Left hemisphere.—In figs. 14, 16 and 18, are respectively shown photographs of the posterior part of the left hemisphere from within, from below and within, and from behind, below and within. Further, in figs. 15, 17 and 19, which exhibit the same views as drawings of two diameters, is shown the exact distribution of the left visuo-sensory area. In all these figures the various numbers are introduced as marks of identification for corresponding regions and fissures. These numbers serve no other purpose and are not the same as those employed in the illustrations of the right hemisphere.

The fissural pattern on the left side is conventional, with the exception of the fact that the posterior cuneo-lingual annectant (situated at 4) is complex and partly superficial, and thereby complicates the posterior extremity of the calcarine fissure.

The visuo-sensory area occupies the strictly normal distribution. It begins anteriorly on the lower lip of the stem of the calcarine fissure, spreading upwards over the apex of the cuneus and downwards towards the collateral fissure (14). Superiorly, it follows the line of the parallel cuneal sulcus (11) (figs 14 and 16), and then that of the commencement (8) and the completion (17) of the superior polar sulcus (fig. 18). It does not, however, occupy the whole of the gyrus enclosed by this sulcus. Here, therefore, the visuo-sensory area is slightly less extensive than usual, or, equally probably, the part of the fissural system 8, iii, 17, where "iii" is placed (fig. 18), represents the sulcus lunatus of Elliott Smith, in which case no marked decrease of extension exists. Inferiorly, the edge of the visuo-sensory area follows the line of the collateral fissure (14) at first, and then, in a quite normal manner, guided by the surrounding sulci, of which 13, 13*a* and 10 (figs. 14 and 16) represent the inferior polar sulcus, it encircles the extremity (marked 4, 7, 6 and 5) of the calcarine fissure (figs. 14 and 16). The fact that the edge of the visuo-sensory area does not reach throughout the gyrus encircled by the inferior polar sulcus indicates a slightly deficient extent of the area, and thus resembles its distribution round the superior limb of the posterior extremity, where it does not occupy the whole gyrus encircled by the superior polar sulcus 8, iii, 17.

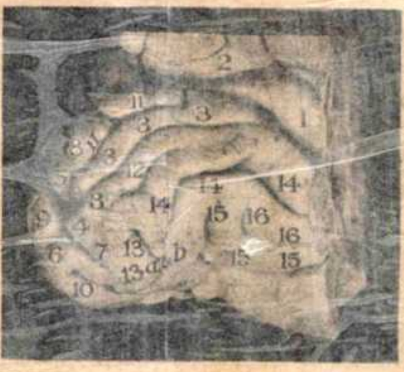


FIG. 14.—Photograph of the left occipital lobe from within. The various numerals are inserted as marks of identification for corresponding regions and fissures in figures 14-19. 1 lies above the stem of the calcarine fissure; and 3 indicates its course, 4 its point of division, and 5, 6 and 7 the portions into which it divides. 5 represents the external portion which appears behind the pole as "=" on fig. 11. 11 lies above the parallel cuneal sulcus, and 14 along the line of the collateral fissure.

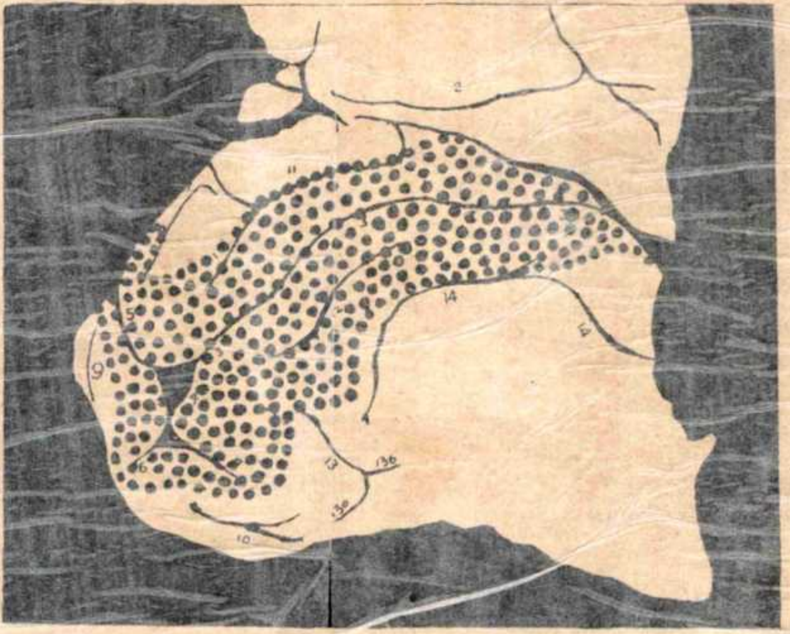


FIG. 15.—Drawing of fig. 14, enlarged two diameters. The numerals correspond with those of the last figure. The visuo-sensory area, in its superficial extension, is indicated by dots. Its normal fissural relationship is well shown. Inferiorly it does not extend as far as the composite inferior polar sulcus 13, 13a, 10.

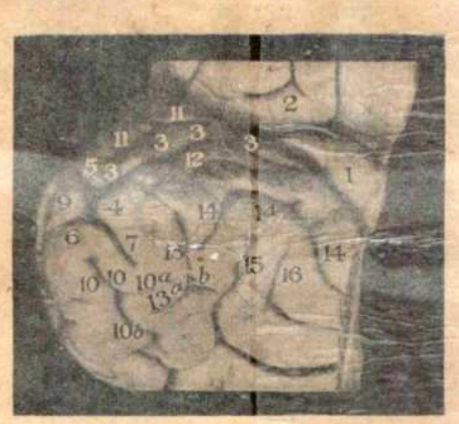


FIG. 16.—Photograph of the left occipital lobe from below and within. The numerals correspond with those in the last figures.

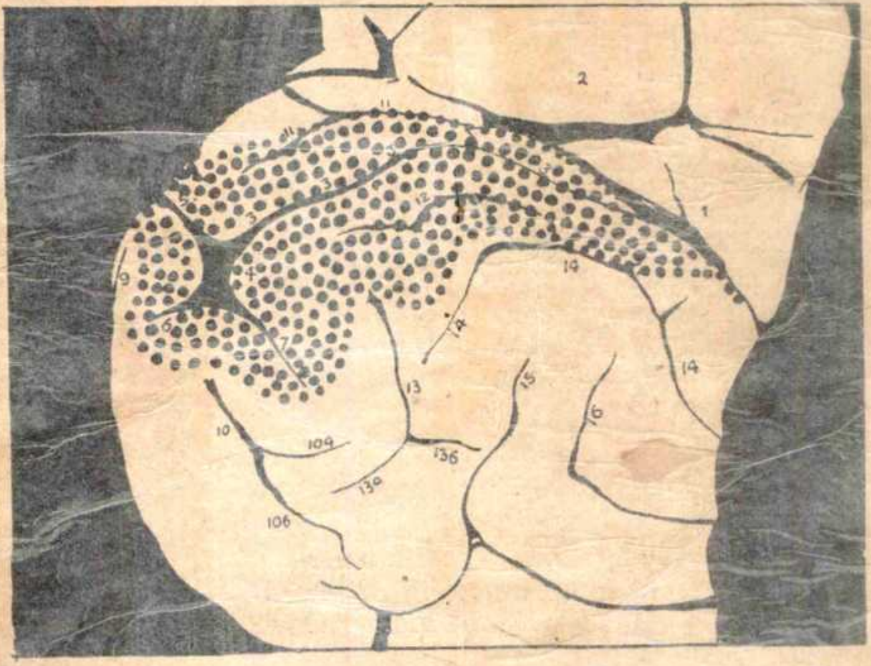


FIG. 17.—Drawing of fig. 16, enlarged two diameters. The visuo-sensory area is better seen in this drawing, and its surface extension in relation to the calcarine stem (below 1), the collateral fissure (14), and the inferior polar sulcus (13, 13a, 10a, 10), is definitely shown.

Right hemisphere.—In figs. 20, 22 and 24, are respective photographs of the posterior part of the right hemisphere fro from below and within, and from behind, below and within. In figs. 21, 23 and 25, which exhibit the same views as drawin diameters, is shown the exact distribution of the right visu area. The various numbers on these figures are introduced of identification solely, as were different numbers in figs. 15, 17 the case of the left hemisphere.

The fissural pattern on the right side is less conventiona the left. The normal truncated appearance so often seen in occipital lobe is accentuated, and the calcarine fissure is muc than its fellow. In one interesting point, however, a resemblance between the two calcarine fissures exists, nar complex posterior extremity (4, 12, 11) associated with the lar superficial and complex posterior cuneo-lingual annectant which should be compared with the corresponding posterior e of the left calcarine fissure (figs. 14, 16 and 18.)

The sulci surrounding the calcarine fissure on the right very aberrant. In association with the great width of the parallel fissures (14 and 15) exist (figs. 20, 22 and 24), w at right angles to the site of the usual parallel cuneal sulcus, sign of which is apparently the T-shaped mesial extremity of fi Inferiorly, the collateral fissure (5) is normal in distribution, bu 6, 9 and 10, and even 13, are all unusual. Fissure 9 en superficially, however, in the calcarine fissure itself between 1 (fig. 24). The posterior branch of 13 possibly represents, a large polar sulcus, but this is uncertain as both these run into the lesion, which, however, just escapes the tip of carine fissure (fig. 24, and also fig. 25).

Allowing for these fissural abnormalities, the distribution ar of the, however decidedly small, visuo-sensory area of the right normal. These, being indicated in the diagrams (figs. 21, 23 need not be described in detail. It may, however, be remarked area extends well forwards along the lower lip of the stem of carine fissure in an absolutely normal manner (fig. 23).

HISTO-PATHOLOGY.

Before dealing with the visual cortex, it may here be r that the infra-cortical visual apparatus is normal.

The *optic nerves* contain abundant nerve-fibres throughout

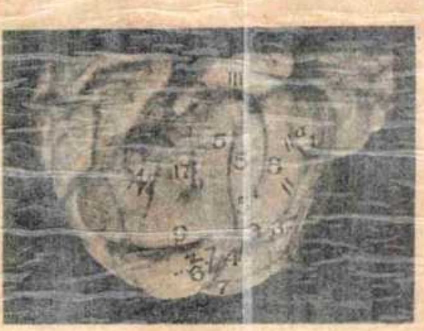


FIG. 18.—Photograph of the left occipital lobe from behind, below and within. The numerals correspond with those of the preceding figures. The letter N is placed just below the inferior extremity of the lesion on this side.

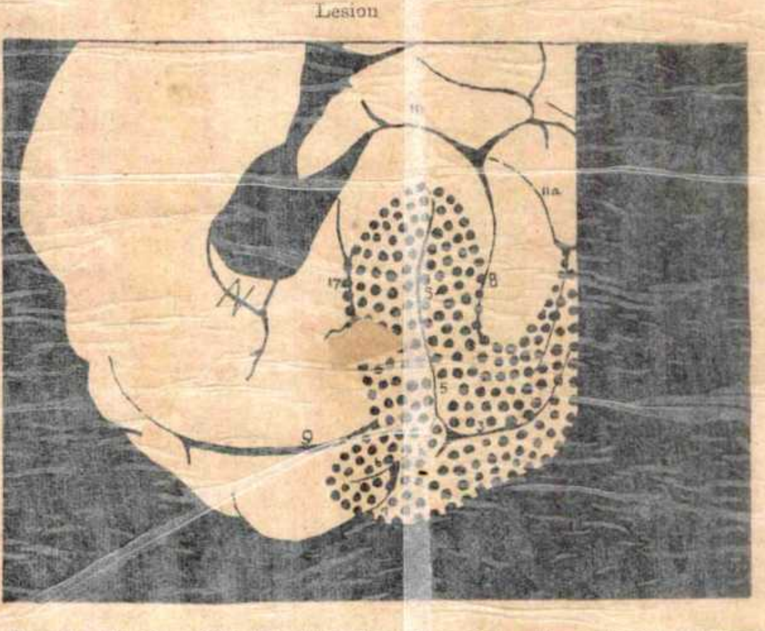


FIG. 19.—Drawing of fig. 18, enlarged two diameters. The posterior extremity of the visuo-sensory area, especially that behind the region of the posterior cuneo-lingual annectant (situated at 4) is clearly defined, as is the relationship of the top of the area to the well-defined superior polar sulcus (17, III, 8). The large black area above the letter N represents the lesion on this side, and the figure thus clearly indicates that the visuo-sensory area is intact in its entirety.

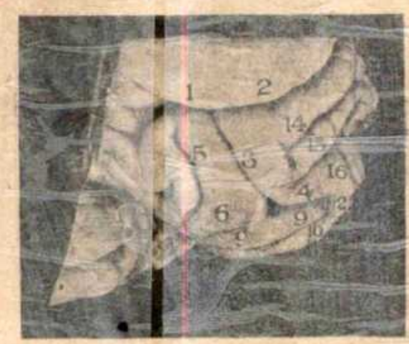


FIG. 20.—Photograph of the right occipital lobe from within. The various numerals in figs. 20 to 25 indicate corresponding points in these figures and thus serve a similar purpose to the numerals employed in the previous set of figures. 1 lies above the calcarine stem, and 2 above the parieto-occipital fissure. 3, 4, 12 indicate the course of the calcarine fissure. 5 indicates the collateral fissure, and the inner ends of 14, 15 the aberrant cuneal sulcus.

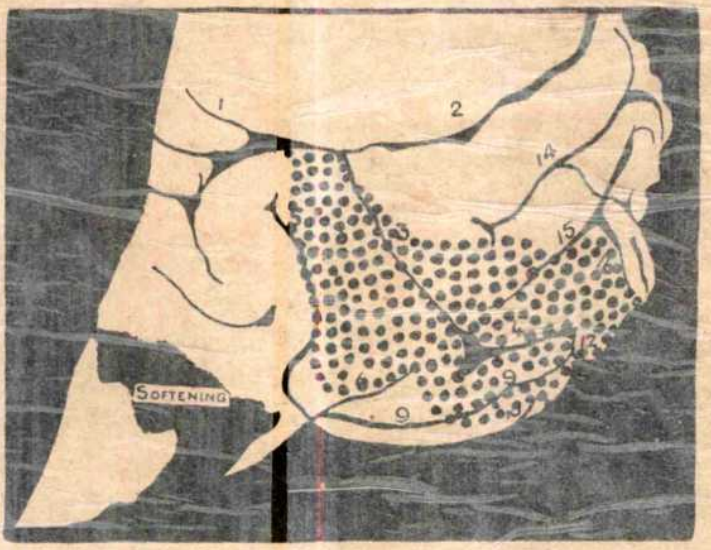


FIG. 21.—Drawing of fig. 20, enlarged two diameters. The numerals correspond with those of the last figure. The visuo-sensory area, in its superficial extension, is indicated by dots. The word "softening" is placed over a local lesion in the left of the figure. The fissuration of this side is rather aberrant, and it should be noted that a similar remark applies to the distribution of the visuosensory area, though to a much less extent.

pared, however, with normals they show some irregularity in shape and a considerable decrease in the size and number of the normal subdivisions of fibrous tissue, together with much fibrosis of the vessels.

The utmost that can be said, from the aspect of morbid histology, is that they exhibit what would be expected after prolonged disuse in the absence of neuronic lesions.

The optic radiations are normal, and the cortical lesions nowhere approach these.

The cortex[1] of the *visuo-sensory* area exhibits striking characteristics. That quite adjacent to and within the calcarine fissure is, as far as can be determined histologically, normal. The whole of the peripheral portion is much thinned, in a more or less patchy manner, but the general characteristics are those of a simple disuse atrophy involving the outer cell or pyramidal lamina (L. II) especially, but to a considerable extent also the outer granule layer (L. IIIa) and the outer fibre or superficial lamina (L. I).

This appearance extends throughout the area, with the result that in general terms the pear-shaped visuo-sensory area consists roughly of a calcarine core of practically normal cortex and of a more or less atrophied surface extension.

This general description applies to both sides of the brain and, as far as can be ascertained, to an equal degree in both.

On the right side, however (see figs. 21, 23 and 25), the area is smaller than on the left (figs. 15, 17 and 19), and its posterior extremity (though not the calcarine fissure) is involved in the lesion (fig. 25).

The surrounding visuo-psychic cortex exhibits no abnormality beyond a slight and variably distributed degree of disuse atrophy of L. II, the outer cell or pyramidal lamina.

CONCLUDING SECTION.

The fact that the visuo-sensory area generally is normal in distribution makes it certain that the gross contraction of the visual fields which is found clinically is due to an associative block, and that this block is not quite so complete on the left side of the visual apparatus as on

---

[1] The cortex of the visuo-sensory area consists of the following layers :—

L. I. *Outer fibre lamina*, or superficial layer.
L. II. *Outer cell lamina*, or pyramidal layer.
L. III. *Middle cell lamina*, consisting of the following layers : L. IIIa. Outer layer of granules. L. IIIb. Line of Gennari or "optic" fibre layer. L. IIIc. Inner layer of granules.
L. IV. *Inner fibre lamina*, or inner line of Baillarger.
L. V. *Inner cell lamina*, or polymorphic layer of nerve-cells.

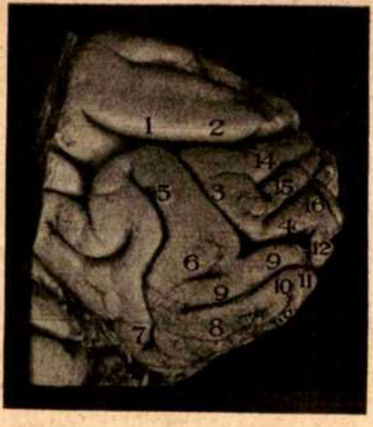


FIG. 22.—Photograph of the right occipital lobe from below and within. The numerals correspond with those of the last two figures.

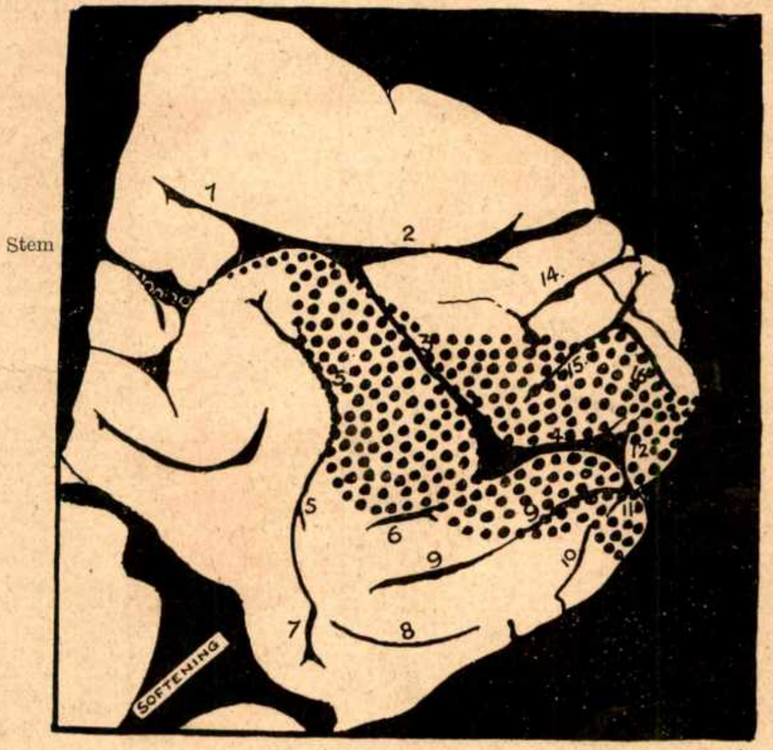


FIG. 23.—Drawing of fig. 22, enlarged two diameters. The general distribution of the visuo-sensory area is better seen in this figure than in fig. 21, and it should be noted that the area extends forwards, as is shown by the white rings, almost to the line of the cross-section at the point where the word "stem" is placed. It is thus incidentally evident, as the fissuration shows, that 1 is in this figure placed not above the calcarine stem, but above a small sub-fissure considerably above it. The word "softening" is again placed over a local lesion distinct from the main softening.

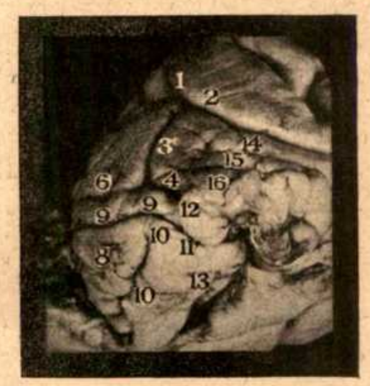


FIG. 24.—Photograph of the right occipital lobe from behind, below and within. The numerals correspond with those of the last figures.

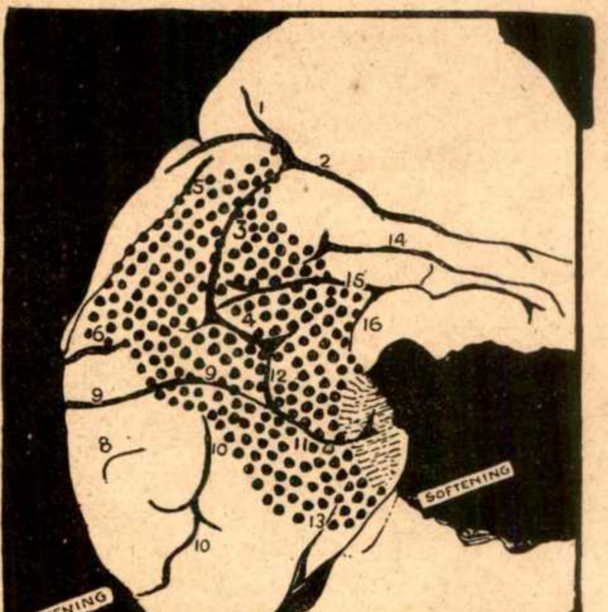


FIG. 25.—Drawing of fig. 24, enlarged two diameters. The word "softening" on the left refers to the small lesion shown in figs. 23 and 21. The same word on the right indicates the posterior limits of the important triradiate lesion already illustrated in general view in fig. 11. The hinder part of the visuo-sensory area is well shown in this figure (line 3, 4, 12, 11). Beyond 11, just at the tip of the calcarine fissure, the first indications of softening begin, but the visuo-sensory area is histologically recognizable right up to the edge of the black area of softening.

the right (and in the right fields of vision as in the left), in association with a smaller or less important lesion on the left side of the cortex. This is indicated histologically by the disuse atrophy of the visuo-sensory cortex which is everywhere marked, with the exception of the more central (calcarine) portion.

It would therefore appear that the calcarine core of the visuo-sensory area serves in some degree as an anatomical basis for macular as distinct from non-macular or panoramic vision.

This conclusion is supported by the fact that the lesion in the right hemisphere reaches up to the posterior portion of this calcarine core. This fact at the same time incidentally explains why in the case the right field of (central) vision was a little more extensive than the left.

It appears, therefore, to be probable that human macular vision is an evolution dependent on (1) the development of binocular vision, i.e., on the employment of corresponding parts of the retinæ simultaneously; and on (2) the simultaneous development of the capability to pay prolonged and individual attention to particular points of the general visual panorama.

Macular vision is thus superposed on the neuronic apparatus for panoramic, and the two types shade into one another, the central parts of the retinæ and the corresponding central (calcarine) cores of the pear-shaped visuo-sensory areas being the parts concurrently employed in macular vision.

The associative block caused by the lesions in this case, therefore, being greater on the right side than on the left, must thus be regarded as having been enough to stop the passage of the feebler panoramic stimuli without interfering seriously with the macular and stronger visual stimuli.

The continued passage of the latter has prevented the onset, in the calcarine core of the visuo-sensory area, of the disuse atrophy which elsewhere in these areas is general.

Lastly, the slight maiming of the right calcarine core posteriorly is responsible for the fact that the clinical visual field of the case was rather less extended on the left side.

The histological investigation of this case may thus claim not only to have explained the clinical features present during life, but to have added to our knowledge of the part played by the visuo-sensory area in macular and in non-macular or panoramic vision. It may, in fact, be stated that the anatomical basis of the former is the cortex of the calcarine core of the pear-shaped visuo-sensory area, and that the anatomical basis of the latter lies in the surrounding and remaining visuo-sensory cortex.

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(c) Anastomotic filaments uniting the temporo-facial branch of the seventh with the auriculo-temporal branch of the fifth. As the facial nerve splits up into its terminal divisions in front of the ear, small anastomotic filaments pass to the neighbouring auriculo-temporal branch of the fifth. As the auricular branches of this nerve pass to the skin of the tragus, superior portion of the helix, and the anterior portion of the auditory canal and tympanum, it would be possible for sensory fibres of geniculate origin to reach the external surface of the pinna by this route. Some sensory fibres may also course with the motor filaments of facial origin which supply the minute subcutaneous muscles of the auricle.

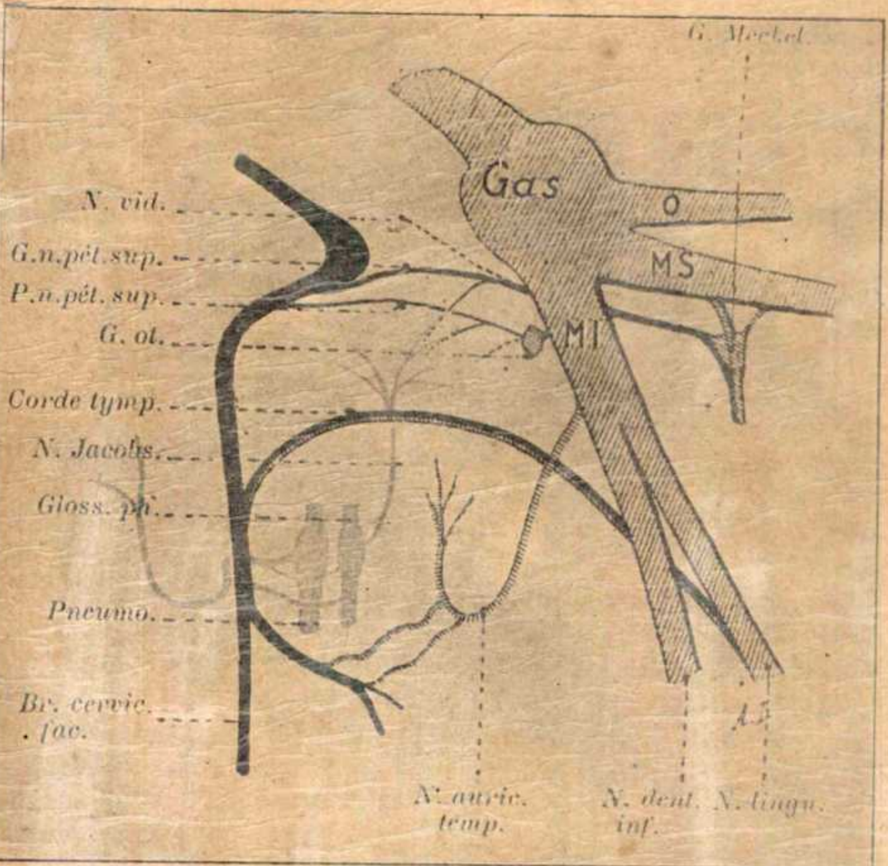

FIG. 9.—From Testut, "Anatomie humaine," 1905, vol. iii. A schematic representation, showing the anastomoses of the facial with the trigeminal, and the auricular branches of the glosso-pharyngeal and pneumogastric nerves.

That cutaneous fibres of geniculate origin should reach the external ear by way of the petrosal nerves and their communications with the trigeminus is, on phylogenetic grounds, very unlikely. It is especially the communications between the facial and the auricular branch of the vagus, the posterior auricular nerve, and the anastomoses with the auriculo-temporal branch of the fifth, to which I would direct attention as the probable pathways of these sensory filaments.

In the classical text-book descriptions of the external auditory canal and the outer surface of the tympanic membrane, the nerve supply is divided between the auricular branches of the trigeminus and the auricular branch of the vagus, the trigeminal area occupying approximately the anterior half of the canal and tympanic membrane, whilst the vagus is accorded the posterior strip. In my descriptions of the extent and distribution of the zoster zone of the geniculate, it was pointed out that the facial in some cases retains a remnant of innervation within the auditory canal and on the typanum. While the zoster evidence in favour of this view is by no means conclusive, it is nevertheless suggestive and should be considered in anatomical investigations in this subject (Wilson [47]). It would be strange indeed if the seventh nerve had no sensory remnant in a domain which was formerly its own (first branchial cleft), while the auricular filaments from the ninth and tenth were still represented in no inconsiderable degree. Indeed, were it not for the traditional anatomical point of view, founded essentially on the dissection method and the motor symptomatology of the seventh nerve, one's first thought would have been of the geniculate ganglion in connexion with the innervation of the structures developing from the hyoid arch.

Intra-oral representation of the geniculate ganglion.—That in man sensory remnants should still persist in what was formerly an important visceral innervation of the seventh within the buccal cavity is not surprising. The herpes zoster evidence indicating the presence of such rudiments consists in the occasional occurrence of an eruption limited to the anterior two-thirds of the tongue in the distribution of the chorda tympani, and on the palate in the region of the anterior pillar of the fauces. Cases of this kind were analysed in my earlier contributions and more recently similar observations have been reported by Palmer [34] and Closier [6]. In Palmer's case (see table) the eruption was distributed on the concha, the posterior wall of the canal, the anti-helix and fossa of the anti-helix, i.e., in the geniculate auricular zone; but in addition there was also an eruption on the anterior pillar of the fauces and the adjacent region of the soft palate. The herpes was accompanied by facial palsy and acoustic nerve symptoms. An almost identical case is reported by Closier, the herpetic eruption on the ear appearing on the concha, tragus, external meatus, and the posterior wall of the canal, but in addition herpetic vesicles were also present on the soft palate. This case, like the preceding, was associated with facial palsy and auditory symptoms. It is, therefore, probable that the geniculate retains in some cases a remnant of its former sensory function within the buccal cavity, in the area of distribution of the chorda tympani and in the region of the soft palate. It will be recalled that many years ago Bernhardt pointed out the rare occurrence of hypæsthesia in the chorda distribution in cases of facial palsy, an observation which was subsequently confirmed by Harvey Cushing [7] in his studies of the trigeminal zone; these observations confirming by another method and in a different group of cases the conclusions which I had reached in regard to the intra-oral visceral remnant of the seventh nerve.

In explanation of the palatal herpes and its possible relation to the geniculate ganglion there would seem to be some anatomical basis in the fact that the lingual branch of the seventh, which innervates the stylo-glossus and palato-glossus muscles at the base of the tongue, also sends mucous filaments to the anterior pillar of the fauces and adjacent region. And, while it is true that this lingual branch has an anastomosis with the glosso-pharyngeal, there is no reason to assume that these mucous filaments are not a part of the sensory facial system and originating in cells of the geniculate ganglion. It is also possible that sensory fibres of geniculate origin pass to the oral cavity by way of the petrosal nerves and their connexions with the trigeminus.

The geniculate ganglion, therefore, in addition to its taste function, has a variable and vestigial intra-oral sensory representation, as have the glosso-pharyngeal and vagus nerves; and all three of these branchial nerves, the seventh, ninth and tenth, are represented both within the mouth cavity and on the external ear.

CHAPTER V.—COMPARATIVE ANATOMY AND ITS BEARING ON THE CUTANEOUS ZONE OF THE FACIAL NERVE.

Of great importance in the final elucidation of this problem of the cutaneous innervation of the facial nerve will be the work of the comparative anatomist. A large literature already exists on the subject of the comparative morphology of the cranial nerves, and the separation and description of their various nerve components by the comparative method. One of the fruits of such investigations has been the demonstration in man of the *nervus terminalis*, which had been traced successively through many of the lower forms. (Johnston [24]). Even as low in the verbetrate scale as fishes, the *nervus terminalis* is vestigial. I mention this instance of the curious persistence of a rudimentary cranial nerve in man as bearing upon the subject under discussion. It is my belief that the cutaneous component of the seventh nerve may also be traced upward through the vertebrate scale by investigation in the field of comparative neurology. At the present time the tendency has been to deny the existence of a cutaneous component of the seventh nerve in forms above the *cylostomes*, notwithstanding the fact that cutaneous fibres are found in the seventh nerve of selachians, teleosts and amphibians, but are usually supposed to originate from the neighbouring sensory systems of the fifth and tenth nerves. One reason for this point of view, I believe, is the traditional anatomical conception that the external ear is innervated by the trigeminus anteriorly, the cervical nerves posteriorly, with a small intervening field of the glosso-pharyngeal and vagus, so that it was very easy and natural to regard the few cutaneous fibres in the seventh nerve as accessions from the trigeminal or vagal systems. Already, however, there are indications of a change from this point of view.

In a recent study of "The Cranial Nerves of Siren Lacertina," H. W. Norris [33], in the chapter entitled "The General Cutaneous Component of the Facial Nerve," writes as follows: "The presence of general cutaneous fibres in the seventh nerve of amphibians has been commonly recognized, but no distinct route of the facialis entering the spinal fifth tract has been found. It is generally assumed by students of nerve components that any and all such fibres found their way into the seventh nerve from the tenth nerve through the ramus communicans cum facialis. To the writer's knowledge no one has found general cutaneous fibres in the root of the facial nerve. Any suggestion therefore of the occurrence of such fibres challenges contradiction and calls for a most critical examination of the so-called evidence." Norris then proceeds to describe such a cutaneous component in *siren*, and his conclusions are as follows: "The apparent occurrence of the general cutaneous constituent in the facial nerve roots also suggests that it is possibly more generally present in other *urodela*, but because of its minuteness and close association with other components has hitherto escaped unnoticed. Its presence is best explained on the ground of a persistence of a function and at one time more fully developed characteristic of the facial nerve."

Judson Herrick [17], in a study of the medulla oblongata of larval *amblystoma*, in a chapter describing "General Cutaneous Roots of the Fifth, Seventh and Ninth Nerves," says: "In a number of longitudinal sections by the methods of Cajal and Golgi I have seen evidence of a few fibres from the seventh root complex entering the spinal fifth root. In one Golgi impregnation such a fibre is clearly defined for a long distance. This fibre bifurcates into ascending and descending branches, both of which enter the spinal fifth root. These fibres probably represent the general cutaneous component of the facialis described by Norris in *siren*, though I have not succeeded in following them peripherally, or recognizing their ganglion, and my observations are so few as to require further control."

Kingsbury [26], as far back as 1897, demonstrated the central connexions of the cutaneous component of the seventh nerve in ganoid fishes, and it is not unlikely that in the near future more anatomical evidence of this nature will be forthcoming, and will confirm the results obtained in man by purely clinical methods of study.

CHAPTER VI.—ON DISTURBANCES OF SENSIBILITY IN THE SENSORY FIELD OF THE FACIAL NERVE.

Conchal hypæsthesia.—The disturbances of sensibility in the geniculate zone after lesions of the facial nerves are slight and comparatively insignificant, because of the small area involved, its rudimentary character, and the overlap from the neighbouring systems of the fifth, ninth and tenth, and upper cervical ganglia. It is also probable from the herpetic manifestations that anomalies and variations in size of the geniculate area are not uncommon. In my earlier contributions to the sensory system of the seventh nerve, attention was called to an area of hypæsthesia in the concha of the ear which was sometimes demonstrable in recent cases of facial paralysis. As was stated at that time, "this disturbance of the sensibility in the conchal region never reached the degree of anæsthesia. The tactile sense was merely diminished or obtunded in this area, and care was required to demonstrate its presence." Since that time I have repeatedly demonstrated the existence of this small area of conchal hypæsthesia in recent cases of facial neuritis, and occasionally after herpetic inflammations of the geniculate ganglion. This area of hypæsthesia, however, is not permanent and disappears early, and cannot be sharply outlined, as in trigeminal anæsthesia; nor, indeed, was this to be anticipated, considering the small size of the area involved, and its interlacement with adjoining sensory zones.

Kidd in his remarks on the cutaneous zone of the seventh nerve makes this statement: "As a matter of fact, if there were any facialis cutaneous fibres in man, we should find in every case of Bell's palsy as complete a cutaneous anæsthesia of the alleged auricular zone as any clinicians have found on the trigeminal cutaneous area after a complete Gasserianectomy." But this surely is not necessary, nor even probable, and had Kidd been familiar with my later work on the zoster zones of the geniculate, glosso-pharyngeal and vagal ganglia [21], which is not mentioned in his article, this statement might have been considerably modified.

Of special interest from its bearing on the sensory disturbances of the external ear is the case of geniculate otalgia reported by Clark and Taylor [2], in which a cure was effected by section of the nerve of Wrisberg. Kidd, who has utilized this case in his discussion of the cutaneous zone of the seventh nerve, approaches this part of his criticism as follows: "And yet, amidst all the well-deserved rejoicings that followed the recital of this case on this New York joy-day, in which Ramsay Hunt took part, actually no speaker asked the question as to what was the post-operative state of cutaneous sensibility on the auricle, and there is even no mention as to whether it was ever tested!" Here again, Kidd indulges in a rather rash statement which is based on an incomplete knowledge of the literature of the subject which he is discussing. The occasion to which he refers was a *later* presentation of the patient to the New York Neurological Society, November 9, 1909 (Clark and Taylor [1]), and which was some months after the original presentation to the American Neurological Association in May, 1909. The case was reported in full in the *Journal of the American Medical Association*, December 25, 1909 (Clark and Taylor [2]), in which the following description of the sensory examination was given: "All sensory examination of the face and external ear proved negative three days after operation. The former area of pain seemed to the patient to be a little less sensitive in the test." So that in this case, while there was no well-defined sensory disturbance, there was a lessened sensibility in the auricular area which had been the seat of pain and which harmonizes with the slight disturbances of sensibility demonstrable after other lesions (seventh palsy and ganglion inflammations) and is in accord with the rudimentary and variable character of the geniculate zone.

Hypæsthesia on the postero-mesial surface of the auricle and adjacent mastoid.—It is occasionally possible to demonstrate in conjunction with conchal hypæsthesia a diminution of sensibility on the postero-mesial surface of the auricle and adjacent mastoid region in cases of recent facial palsy. This area corresponds to the auricular distribution of the vagus and that of the ascending filament of the posterior auricular branch of the seventh nerve, which, according to Valentin, carries cutaneous filaments to this region. A hypæsthesia in this region may be caused either by involvement of the sensory filaments of the seventh nerve, or of the auricular branch of the vagus as it crosses the facial at the lower end of the Fallopian aqueduct.

As has already been stated, this area is sometimes the seat of herpes in cases of geniculate inflammation, and it is probable that the seventh and tenth ganglia both have rudimentary representations in this post-auricular strip (see fig. 4).

Sensory disturbances of the face and neck in cases of geniculate ganglion inflammation.—In the interpretation of sensory disturbances in the geniculate zone in cases of herpetic inflammations, it should not be forgotten that in some cases there is an associated hypæsthesia and hypalgesia in the trigeminal distribution and occasionally in the cervical zones as well. This I have regarded as evidence of a mild inflammatory reaction in neighbouring ganglia, and has been fully discussed in an earlier paper in the chapter, "Multiple Involvement of Ganglia in Zona," [22]. Such hypæsthesias are often accompanied by darting pains, loss of the corneal reflex, and groups of aberrant zoster vesicles, all of which are additional indications of ganglionic involvement. It is also well to recall that in cases of facial palsy there is not infrequently a diminution in the sensibility of the face on the paralysed side which has been variously interpreted: as of hysterical or psychogenic origin, as a functional derangement of the ganglion of the fifth nerve induced by motor insufficiency of the paralysed face; as the result of hypotonia of the cutaneous muscles with consecutive feebleness of the dermal papillæ; as a neuritis of peripheral sensory fibres passing from the trigeminus to the facial muscles. It should, however, be emphasized that whatever the underlying cause, psychogenic, physiological, or neuritic, of this mild hypæsthetic disturbance, it should not be identified in any way with the geniculate area.

Intra-oral sensory disturbances of geniculate origin have as yet not been demonstrated, except for the rare occurrence of hypæsthesia in the chorda distribution of the tongue, as was shown by Bernhardt in cases of recent facial palsy. This observation has been confirmed by many since Bernhardt first drew attention to it, and all agree that it is slight and very inconstant.

It is probable that routine examinations will show a similar inconstant hypæsthesia on the palate near the faucial margin, but thus far I have not encountered it (*see* fig. 5).

On two occasions the passage of a sound into the Eustachian tube was less sensitive on the side of an inflammation of the geniculate ganglion with facial paralysis. This occurrence I was inclined to explain by the participation of the sensory system of the geniculate in the innervation of the tympanic cavity and its prolongations, the Eustachian tube and mastoid cells.

CHAPTER VII.—RECAPITULATION AND CONCLUDING REMARKS.

The facial nerve is, therefore, to be regarded as a mixed cranial nerve, homologous with the trigeminal, glosso-pharyngeal, and vagal systems. In common with these, the facial possesses a definite group of symptoms and syndromes directly dependent upon involvement of its sensory system.

In the lower forms of life the sensory facial equals, and may even exceed in importance, the motor functions. In man, however, the conditions are reversed—the motor function is predominant, and its sensory system has retreated before the advance of the trigeminus in front and the cervical areas posteriorly.

In comparative anatomy the facial is the nerve of the first branchial or ear cleft; and evidences of this ancient phylogenetic relationship is still present in man, in the cutaneous remnant which persists on the external ear as well as its relations to the other structures of the auditory mechanism which take their origin from the region of the spiracle or first visceral cleft.

That portion of the facial sensory system which still persists in man is concerned in the innervation of the internal ear, the middle ear, and its prolongations into the Eustachian tube and mastoid cells, and the skin on certain portions of the external ear. There is also evidence that a vestigial remnant occasionally participates in the innervation of a strip on the postero-mesial surface of the auricle and within the buccal cavity in the chorda tympani distribution, and on the palate near the anterior pillar of the fauces.